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is dedicated to the memory of
Dr. Gillette Hayden
for her pioneer efforts and
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of periodontia and the
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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

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Dr. Hunter S. Allen was graduated from Atlanta Southern Dental College in 1918 and has limited his practice to periodontia since 1923. He has been active in organized dentistry, serving as president of the Alabama Dental Association in 1941 and Secretary from 1933-38. He was President of the Southern Academy of Periodontology, 1947-48. He is also a Fellow of the American College of Dentists.

THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED FIFTY-ONE

The Uneven Distribution of Calculus in the Mouth

by S. WAH LEUNG, D.D.S., Ph.D.* *Pittsburgh, Pa.*

A COMMON but unexplained observation concerning the distribution of calculus in the mouth is the finding that the concretions are deposited most frequently and in the greatest quantity on the buccal surfaces of the upper molar teeth and the lingual surfaces of the lower anterior teeth. These surfaces are also in closest proximity to the openings of the ducts of the major salivary glands and, therefore, would presumably be the first to interact with freshly secreted saliva.

This characteristic distribution is not only unaccounted for by the current theories of the mechanism of calculus formation, but indeed, has been cited as evidence against the acceptableness of certain proposed theories.[1, 2, 3]

The results of studies of the behavior of salivary calcium and phosphorus in the presence of added hydroxylapatite suggest a plausible explanation for the peculiar oral distribution of calculus. It has been repeatedly demonstrated that such substances as powdered bone, commercial tricalcium phosphate, powdered enamel, and synthetic hydroxylapatite, when added to saliva, will result in the precipitation of relatively large amounts of calcium or calcium and phosphorus from the saliva.[3, 4, 5, 6, 7, 8, 9, 10, 11, 12] Incidental to studies which we have recently made regarding the behavior of salivary calcium and phosphorus in relation to calculus formation, we noticed marked differences in the amounts of the two minerals which were precipitated from the saliva, depending on the presence of hydroxylapatite. The accompanying table shows that, in contrast to the large amounts of calcium and phosphorus precipitated in the presence of synthetic hydroxylapatite,† aliquots of the same samples, treated identically but without the addition of hydroxylapatite, lost relatively little of the two elements.

Chemical and x-ray diffraction analyses of calculus have shown it to consist, in common with bone, enamel and dentine, of calcium phosphate arranged in

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*University of Pittsburgh School of Dentistry, Pittsburgh, Pennsylvania. The work discussed in this paper was undertaken in the Department of Physiology of the University of Rochester School of Medicine and Dentistry while the author was a Fellow in the Division of Dental Research of that Institution.

†We wish to thank Dr. H. C. Hodge for supplying us with the powdered hydroxylapatite used in these experiments. The salt was found to have a Ca:P ratio of 2.10. Its manner of preparation and the analytical data of its composition may be obtained by referring to: Hodge, H. C., LeFevre, M. L., and Bale, W. F., Ind. Eng. Chem., Anal. Ed., 10:156, 1938. It is identified as sample H-5.

*Effect of Treating Saliva with Powdered Synthetic Hydroxylapatite (H.A.)**

	Calcium mM		Phosphorus mM	
	Sample 1	Sample 2	Sample 1	Sample 2
Control	1.83	1.62	4.62	4.54
5% CO_2, no H.A.	1.25	1.12	4.15	4.73
5% CO_2, with H.A.	0.51	0.53	3.05	3.82

*The 3 aliquotes of each sample were sealed in air tight vessels. Nothing was added to the control. To one of the experimental specimens was added a gas mixture containing 5% CO_2, to the other 5% CO_2 plus powdered hydroxylapatite. All specimens were continuously rotated for 5 days at a temperature of 37°C. They were then centrifuged and the supernatants analyzed for Ca and P.

the hydroxylapatite crystal lattice structure. It would be reasonable to expect, therefore, that calculus should affect salivary calcium and phosphorus in the same manner as synthetic hydroxylapatite, namely, to enhance their precipitation from saliva. One should also expect the greatest effect to occur in those regions of the mouth supplied with an abundant quantity of freshly secreted saliva relatively rich in calcium and phosphorus. These are the same areas in which calculus has been found to be most heavily deposited. It is our contention, therefore, that the heavy incrustrations of calculus found on the abovementioned tooth surfaces are the result of the action of the initially present calculus on the calcium and phosphorus of the saliva. It will be noticed that this hypothesis, which explains the presence of heavy calculus formation on the tooth surfaces in closest proximity to the openings of the salivary ducts, supplements, but is independent of, and offers no solution to, the problem of how the formation of the initial small amount of calculus is brought about. In its present form it neither supports nor replaces any of the existing theories of calculus formation. It is believed that, perhaps by one or more of the several mechanisms suggested in the literature, small quantities of calculus are initially deposited on many of the tooth surfaces. This calculus then acts to induce further (and probably more rapid) deposition from the saliva, simultaneously with the continued production of calculus by the first method. But, since the calculus present on the tooth surfaces contiguous to the duct orifices encounter calcium- and phosphorus-rich saliva first, the effect here will be greatest. The partially depleted saliva, passing onto other tooth surfaces, will be able to supply little calcium and phosphorus to them. Hence, the amount of calculus there will be less.

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Current Problems in Terminology and Classification of Gingival and Periodontal Disease*

By ISAAC SCHOUR, D.D.S., PH.D.**

INTRODUCTION

CLASSIFICATIONS and terminology become more exact as our knowledge progresses. In the field of periodontology the all-inclusive term periodontal disease denoting a variety of lesions of the periodontal structures (gingiva, alveolar bone and periodontal membrane) is too diffuse. It has become necessary to use more precise and accurate terms in order to designate both the particular structure involved (gingiva, alveolar bone and periodontal membrane) and the particular pathologic process which predominates (inflammation, degeneration, etc.).

Several modifications and terms are suggested in the light of recent studies to provide for greater accuracy and precision. It is fully recognized that further improvements will be achieved as our knowledge of periodontal structures and of their diseases advances.

The broad division of periodontal disease into gingivitis, periodontitis and periodontosis has withstood the test of time. These terms are useful for general description but should be supplemented with more precise language to designate the particular structures affected. This can be done (Table 1) even though our current knowledge is incomplete, especially in respect to etiology and pathogenesis.

Greater discipline in terminology could be achieved by employing anatomical subdivisions or by indicating the age period or particular state of health when these factors appear to be of significance.

It must be pointed out that increased precision in terminology should be carefully guarded, lest it be employed to ascribe specificity which is not justified on the basis of our present knowledge. Most periodontal diseases are nonspecific in character. Thus, a microscopic examination of a gingival biopsy will indicate the basic pathologic disturbances (gingivitis or gingivosis) but in most instances will not permit a recognition of the specific etiology. The possible exception is acute leukemia where the gingival lesion will show the basic hematologic picture which is characteristic of the disease (Orban, et al., 1948; Wentz, Anday, and Orban, 1949).

Thus, the term gingivitis in pregnancy is to be preferred to pregnancy gingivitis, since the great majority of cases of gingivitis in pregnant women are identical with gingivitis in non-pregnant women or other individuals (Maier and Orban, 1949). Similarly gingivitis or periodontitis *in* diabetes is more appropriate than diabetic gingivitis (Ray and Orban, 1950).

GINGIVAL DISEASE

The tendency to use gingivitis as synonymous with gingival disease should be corrected. In this report, gingival diseases will be considered under two groupings: *gingivitis* and *gingivosis*.

GINGIVITIS

Recent efforts to develop a quantitative method of assessing gingival disease (P-M-A method) (Schour and Massler, 1947, 1948) have led to a renewed emphasis of

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*Abstract of discussion presented before Symposium on Periodontal Disease, May 19, 1950, Cleveland, Ohio.

**From the Department of Histology, University of Illinois, College of Dentistry. This report is based on discussions and findings that have developed during the course of investigations on the epidemiology and histopathology of the gingiva which were supported (in part) by research grants from the Division of Research Grants and Fellowships of the National Institute of Health, United States Public Health Service.

the anatomical classification of the gingiva in terms of its three component portions: papillary, marginal, and attached gingiva.[1] This gives rise to the more precise designations of *papillary gingivitis, marginal gingivitis* and *attached gingivitis* depending on which region is predominantly involved in a given case (Schour and Massler, 1947). For example, where the inflammation of the marginal gingiva is the predominating characteristic, marginal gingivitis is more accurate than just gingivitis.

The anatomical subdivisions of gingivitis has proved to be a clinical aid not only to the epidemiologic approach but also to the routine clinical examination of the oral cavity (Orban, 1948).

GINGIVOSIS

The term gingivosis has been used to denote the type of gingival disease in which degenerative changes of the connective tissue predominate (Schour and Massler 1947). Thus gingivosis bears the same relation to gingivitis that periodontosis holds to periodontitis. To employ the term gingivitis for all types of gingival lesions would be just as inaccurate and misleading as to employ the term periodontitis for all types of periodontal lesions.

A critical analysis of gingival lesions observed in institutionalized children suffering from nutritional deficiencies (especially in Vit. B. Complex), led to a recognition of a degenerative change in the gingiva which appeared to correspond to a comparable breakdown of the periodontal membrane in periodontosis. The term gingivosis thus appeared to be more appropriate and accurate than the customarily all-inclusive term gingivitis.

Recent histochemical studies of the connective tissue changes in "Desquamative Gingivitis" have indicated that the primary involvement in this lesion is a degenerative breakdown and depolymerization of the cementing substances of the connective tissue and particularly of the basement membrane (Engel, Ray and Orban, 1950). Orban has more correctly classified this disease as "Desquamative Gingivosis."

PERIODONTAL DISEASES

The term periodontal diseases has been used loosely to include gingival disease as well as disease of the other supporting structures. The subdivisions under periodontal disease of *periodontitis* and *periodontosis* are generally accepted and have contributed toward better diagnosis and treatment. Further clarity, however, may be attained by further subdivisions to indicate whether the gingiva, the alveolar bone, or the periodontal membrane is primarily involved.

PERIODONTITIS

Weinmann (1941) has shown on the basis of a detailed microscopic analysis of sections of human jaws that the inflammation of the supporting structures of the tooth progresses in successive stages from the gingiva (Stage I) to the alveolar bone (Stage II) and finally if at all to the periodontal membrane (Stage III).

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1. The following definitions are based on Orban's report (1948) on the Surface Characteristics of the Gingiva.

Papillary gingiva: that part of the gingiva that fills the interproximal space between two adjacent teeth, consisting partly of attached and partly of free gingiva.

Marginal gingiva: that part of the free gingiva that is localized at the labial buccal, and lingual aspects of the teeth.

Attached gingiva: that part of the gingiva that is firmly attached to the underlying tooth and bone and stippled on the surface.

Free gingival groove: shallow line or depression on the surface of the gingiva between the free and attached gingiva.

2. Blakiston's New Gould Medical Dictionary defines periodontitis as "inflammation of the periodontal membrane." This definition is incomplete and therefore misleading. In the great majority of cases of periodontitis, the periodontal membrane is only slightly and secondarily affected. (James and Counsell, 1927; Thoma and Goldmann, 1937, Box 1940; and Weinmann, 1941)

TABLE 1. *Classification of Periodontal Diseases*

	Anatomical Divisions		*Inflammatory Diseases*		*Degenerative Diseases*
PERIODONTIUM	GINGIVA	papillary marginal attached	GINGIVITIS ↓	papillary gingivitis marginal gingivitis attached gingivitis	GINGIVOSIS
	ALVEOLAR BONE	periosteum supporting bone bone marrow alveolar bone proper (Lamina dura)	PERIODONTITIS ↓	ALVEOLAR OSTEITIS	PERIODONTOSIS ↑
	PERIODONTAL MEMBRANE	periosteum periodontal ligament pericementum		PERIODONTITIS PROPER	

Arrows show usual direction of the course of the disease from initiation to termination.

Stage I is *Gingivitis*. This lesion was discussed above.

Stage II may be best termed *Alveolar Osteitis*. This stage obtains when the gingivitis advances and follows the course of the blood vessels and spreads into the bone marrow of the subjacent alveolar bone and along its periosteal surface. The pathologic process consists of fibrosis and round cell infiltration of the bone marrow, resorption of the alveolar crest and especially of the cortical compact bone, destruction of the alveolar supporting bone and, finally, of the alveolar bone proper of the alveolar bone.

Stage III may be termed *Periodontitis Proper*. It obtains when the inflammatory process has spread into the periodontal membrane.

It is evident that periodontitis is not synonymous with the inflammation of the periodontal membrane.[2] The general term periodontitis should therefore be supplemented and qualified when the predominating involvement lies in the alveolar bone (*periodontal osteitis*) or in the periodontal membrane (*Periodontitis proper*). These more specific terms offer advantages of increased accuracy in diagnosis which are similar to those derived from specifying papillary, marginal and attached gingivitis rather than just gingivitis.

An example of the value of specifying wherever possible the particular tissue disturbance in a given case of periodontitis is seen in "occlusal traumatism." Gottlieb and Orban and others have shown that the periodontal membrane is the tissue which is primarily affected in experimental and clinical occlusal traumatism. The term "*traumatic periodontitis proper*" therefore expresses best the basic pathologic changes in the particular structure involved.

The subdivision of gingivitis, on the basis of anatomical regions is relatively simple, since the papillary, marginal and attached portions are clinically visible. The subdivision of periodontitis on the basis of anatomical and structural characteristics is more difficult even though the terms *marginal* and *periapical periodontitis* have been useful. In most cases microscopic examination is necessary in order to evaluate and distinguish the inflammation of the alveolar bone and inflammation of the periodontal membrane. It is suggested, however, that careful discipline in roentgenographic examination might permit a recognition of the degree of advance in periodontitis from the first to the third stage.

One basic difference between periodontitis and periodontosis is shown in the arrows in Table 1 (Weinmann, 1941; Orban and Weinmann, 1942). Periodontitis starts in the gingiva, spreads to the alveolar bone

and may finally invade the periodontal membrane. Periodontosis follows a reverse course. It starts in the periodontal membrane and quickly spreads to the alveolar bone.

Table 1 summarizes the terms and classifications discussed in this report.

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The Epidemiology of Periodontal Disease

By C. D. MARSHALL-DAY, B.D.S., D.M.D., M.S., PH.D.,* *Boston, Mass.*

MUCH has been written of periodontal disease in ancient and modern times. It is abundantly clear from the literature on the subject that man has always suffered from the ravages of this disease. In his book, "The Paleopathology of Ancient Europe," Sir Marc Armand Ruffer established conclusively that periodontal disease was the commonest of all diseases revealed in the embalmed bodies of the Egyptians of 4000 years ago.[1] Pierre Fauchard in 1680 wrote extensively about the disease. Indeed, references to periodontal disease can be found in almost every language from the earliest times to the present; and epidemiological surveys carried out in many parts of the world have seldom failed to furnish evidence of disease of the supporting structures of the teeth. Yet we still have much to learn regarding the etiology, pathology, treatment and prognosis of the disease; and singularly little statistically reliable information is available regarding its epidemiological characteristics. This has been particularly evident to those of us who have attempted to carry out surveys of this sort in various parts of the world, and to compare our findings with those of others.

The reasons are not far to seek. The absence of an adequate and uniform classifi-

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cation of periodontal diseases for many years has been an important factor in the confusion that exists. In different countries, and sometimes in the same country, many different classifications have been used or suggested. The classifications of Box in Canada, of Fish in England and of Orban in the United States come to mind. Many of these classifications, though differing in terminology, are essentially the same, at least in so far as the major groupings are concerned. The general acceptance of the recommendations of the Nomenclature Committee of the Academy of Periodontology has done much to secure standardization of terminology in this country.[2]

Probably the chief reasons for the paucity of reliable epidemiological data on periodontal disease are the lack of a satisfactory system for examination and accurate diagnosis, and the absence of uniformity in methods employed in the assessment of the disease in its various forms. It must be recognized also that it is not always entirely possible to recognize incipient disease processes, or to differentiate abnormal changes from 'normal' or anatomical variations in color, contour and texture of the gingivae.

A number of other reasons could be advanced to account for the comparative absence of reliable statistical data on the incidence and prevalence of periodontal disease, and to explain the general lack of appreciation of its widespread distribution. It must be admitted that in the great majority of oral disease surveys so far carried out by the United States Public Health Service and other agencies in this and other countries, particularly in the younger age-groups, roentgenographic examinations have not been included. They are expensive, time-consuming and sometimes impossible to carry out for a variety of reasons; but the result has been that not only has the caries picture been grossly understated through failure to detect interproximal lesions, but also, and perhaps more important, the presence of early periodontal breakdown has remained unrecognized and unreported. Thus it comes about that in the absence of any reliable information to the contrary, the dentist shares with the public the delusion that periodontal disease is a disease of middle age. While not entirely excusable it is at least understandable, particularly in areas of high caries incidence, that the dentist is so overwhelmed by the caries picture that his attention is focused on what can more readily be seen, and thereby diverted from the periodontal area. Indeed in most cases attention has never been adequately directed there; for the teaching of periodontology occupies only 2.10 per cent of the time of the whole dental curriculum.[3] Thus the significance of periodontal disease as one of the two major causes of tooth loss, and the greatest cause after the third decade of life, has never been fully appreciated by the dental student, the dental practitioner or the dental investigator.

In 1948 Chilton[4,5] contacted the various dental colleges in this country to ascertain their views on the epidemiological aspects of periodontal disease. The replies received stressed the almost complete lack of reliable statistical data, and the difference of opinion on the distribution and other epidemiological characteristics of the disease in the American population. It would seem that the very small amount of data available has, for the most part, been based upon impressions only. This absence of information is not confined to this country alone. Data on periodontal disease in its various forms in other countries are just as scanty and, in most cases, equally unreliable.

It is therefore with some diffidence that one approaches this aspect of periodontal disease, or attempts to correlate the available data on the subject. The fact that we have carried out surveys in several different areas, including India, Puerto Rico, the Virgin Islands and the United States, may be of some assistance and some interest; at least it is possible to make some comparisons because of the degree of uniformity in methods used for the assessment of periodontal disease.

TABLE I

Country	Author	Year	Number Examined	Age Group	Per cent with Gingivitis	Group
United States	Brucker[16]	1943	1,492	4-16	8.8	Newark, N. J.
	Messner et al.[15]	1936	1,000,000+	6-14	3.5-8.6	26 States
	McCall[17]	1933	4,600	1-14	98.0	New York
	Day[42]	1948	234	6-18	73.0	Rochester, N. Y.
Great Britain	Campbell & Cook[18]	1942	2,924		2.2	Dundee Hospital
	King[20]	1945	313	12-18	88.0	Dundee
	Ainsworth & Young[43]	1925	4,063	2-15	60	England & Wales
Isle of Lewis	King[44]	1940	1,280	6-15	90	
Sweden	Westin et al.[45]	1937	1,141	school children	86.5	
India	Day & Shourie[35]	1947	1,054	(boys) 9-17	99.4	Low to middle-class school (Lahore)
	Day & Shourie[35]	1947	179	(girls) 9-17	73.7	High socio-economic level (Lahore)
	Day[46]	1944	200	(boys) 13	81	Kangra District (poor nutrition)
	Day & Tandan[47]	1940	756	13	68	Middle class (Lahore)
	Day & Shourie[26]	1944	613	5-15	80	Low-middle class schools
New Zealand	Saunders & Taylor[48]	1938	36	4-14	94	Maori Children
Puerto Rico	Day et al.[40]	1948	1,648	6-18	60-79	Puerto Rican boys and girls
Virgin Islands	Day & Shourie[36]	1950	823	6-18	57	Boys and girls 91% negro population

DISTRIBUTION OF PERIODONTAL DISEASE

Even from the limited and inadequate data available it is apparent that periodontal disease constitutes a serious and universal public health problem. This is true because in some degree and in one form or another it affects large population groups at all ages, and so frequently remains undiagnosed and untreated until the disease is well advanced. When recognized the radical treatment of extraction is too frequently resorted to because of a lack of knowledge of the disease process and the inability to treat it conservatively. When large population groups are affected, periodontal disease becomes a major public health responsibility not only as an important cause of tooth loss and consequent reduction in masticatory efficiency but also because of its probable influence upon general health through focal infection.[6, 7, 8, 9, 10, 11, 12, 13, 14] For these reasons it is important that reliable epidemiological data be made available.

The entirely unsatisfactory state of our knowledge is underlined by a review of the literature indicating the wide range of prevalence reported by various investigators. Examples may be given for the United States and Great Britain. In the United States Messner and others,[15] following an examination of more than 1,000,000 white boys and girls in various age groups from 6-14 years of age, reported gingivitis prevalence of only 3.5-8.6 per cent. Brucker[16] reported a similar low prevalence of gingivitis, 8.7 per cent, in a group of children

from 4-16 years of age. On the other hand our own unreported observations on a small group of children (234) from 6-18 years of age, in Rochester, New York, showed a 73 per cent prevalence of gingival disease; and McCall reported a prevalence of 98 per cent.[17]

In Great Britain, Campbell and Cook[18] reported a gingivitis prevalence in 2924 cases examined of only 2.2 per cent, while King[19,20] using severe diagnostic criteria found a prevalence of 55-97 per cent. Both of these investigations were in the same area, Dundee, Scotland. It is inconceivable that this great disparity in findings in the United States and Great Britain could have occurred had all investigators used the same diagnostic criteria.

These and other findings on the prevalence of gingival disease in various geographical areas have been reviewed in Table 1. From this review it seems apparent from the wide range in prevalence reported, sometimes in the same area, that these differences cannot be actual, but are related to the lack of agreement in the assessment of gingival disease. It seems equally apparent from the greater degree of agreement among investigators that the higher figures give a more accurate picture of conditions in the groups of children examined.

It is difficult to obtain an accurate estimate of the prevalence of other forms of periodontal disease, particularly those forms associated with alveolar bone destruction, for the reason that comparatively few investigations of an epidemiologic nature have been accompanied by the use of roentgenograms. An analysis by Miller and Seidler[21] of 1003 cases examined roentgenographically, showed a prevalence of approximately 60 per cent. A dental survey of 500 American army air force pilots with the use of roentgenograms disclosed a 29.2% incidence of periodontal disease.[22] Roentgenographic examination of a total of 568 Indians in the Punjab, from 9-60 years of age, showed a very high prevalence of periodontal disease. We established the fact that in the Indian subject periodontal disease, as manifested by actual bone resorption, has its incipiency in childhood, and that after the age of 16 years bone resorption in varying degree is almost universal. Alveolar bone resorption was found in 8% of cases between the ages of 13 and 14 years and in 83% of all subjects between the ages of 13 and 60 years. From 17 years of age all subjects were affected.[23]

Many of the statements found in the literature are based on impressions only, or on clinical evidence without roentgenographic examination. Broderick[24] states that "pyorrhea is unknown in childhood, rare in the teens and twenties and not common under forty years of age." Masuda[25] is of the opinion that periodontal disease occurs infrequently in the mouths of persons under 25 years of age.

In the Punjab province in India a clinical examination without the use of roentgenograms revealed a 62% prevalence of chronic suppurative periodontitis in a group of Indian males below the age of 30 years.[26] Subsequent work gave every indication that the roentgenographic examination would have materially increased these already high figures. There is reason to believe that this extremely high prevalence of periodontal disease in India is related more to nutritional than to racial or geographical factors. Only by a complete study of all possible factors involved and the use of uniform methods of assessment can the effects of race differences be finally evaluated. At present no reliable data are available on the effects of race on the incidence of periodontal disease.

OCCUPATION AND HABITS

Schour and Sarnat[27] list a number of gingival manifestations of occupational diseases. Various classes of metal workers commonly experience inflammation of the oral mucosa. Metal galvanizers or other workers in zinc are reported to develop a characteristic chronic marginal gingivitis resistant to usual forms of treatment. A

characteristic gingivostomatitis has also been observed in the case of chrome platers and in the case of persons whose occupation brings them into contact with corrosive and caustic agents as in acid manufacturing plants and storage battery repair shops. Electric arc welders are said to exhibit a type of gingivostomatitis having many of the clinical features of a fusospirochetal infection. Cement workers, persons employed in caustic potash plants or other individuals whose occupation brings them in close contact with strong alkalis, frequently suffer from inflammation of the mucosa with gingival involvement. A persistent low grade gingivostomatitis has been reported in chemists constantly exposed to chemicals or fumes. Watch dial painters and other radium workers are said to exhibit a higher incidence of gingivitis and periodontitis. Persons exposed continuously to various organic and inorganic dusts and other irritating substances are stated to be particularly susceptible to gingivitis. Chronic mercury poisoning with characteristic soft swollen gingivae is not uncommon in certain occupational groups. At one time there was considerable controversy regarding the possible ill-effects of the mercury used in amalgam fillings and of mercury vapor in dental offices.[28] There is no evidence of harmful effects from either. A recent investigation by Grossman and Dannenberg[29] indicates that the amount of mercury vapor with which the average dentist comes into contact in his office or laboratory is not toxic, but that the concentration of mercury vapor in the air of the dental office is directly proportional to the amount of mercury used by the individual dentist.

Some of these occupational diseases of the gingiva and other oral tissues are due to direct action of the causative agent and some occur as a secondary manifestation of a systemic disturbance. For most of them protective measures are available, and in most cases poor oral hygiene and ill health render the oral tissues particularly susceptible.

Though much clinical evidence has accumulated with reference to oral manifestations of occupational origin, there would seem to be no reliable statistical data available on this subject.

There would appear to be some evidence that certain habits may be an etiological factor in gingival inflammation and in breakdown of the periodontal tissues. The habitual neglect of proper oral hygiene is often an important causative factor. One has the impression, based on considerable clinical experience in India, that the irritation from the habit of chewing betel-nut in India, Ceylon and other Asiatic countries may be an important factor in periodontal breakdown; but once again reliable data are lacking.

Nutritional habits also probably play an important role. Anker Baaregaard[30] reports that the periodontal tissues show marked differences in primitive and civilized natives in Greenland. The primitive Eskimo subsisted on a native diet consisting mostly of protein and fat from meat and fish, and containing adequate vitamins and mineral salts. In one generation the consumption of imported 'civilized' food increased 375 per cent by weight, and from 1908 to 1931 the percentage of food calories in the form of carbohydrates rose from 10 to 50. In the primitive Eskimo Baaregaard reported much abrasion and well-developed periodontium with high resistance. Mild gingivitis was common but periodontitis was rarely seen and periodontosis never. Functional harmony was the rule. In the civilized Eskimo there is little or no abrasion, and weak periodontium with low resistance; and the incidence of gingivitis is 100 per cent. Though periodontosis is not seen, periodontitis with pockets and bone loss is common. It is of interest that tartar of both the supra- and subgingival types was found to be common in both the primitive and civilized Eskimos but more common in the primitive natives despite the very much lower incidence of periodontal disease.

Another habit which has received some

attention by investigators is the use of tobacco in its relation to periodontal disease. Himling and Pedersen[31, 32] describe a specific type of gingivitis, which they term gingivitis toxica, caused by the use of tobacco, especially chewing tobacco. Lesions involving destruction of the gingiva and bone as far as the root apices of the first and second molars are reported following the placing of chewing tobacco in the buccal fold for prolonged periods of time. Pindborg[33] examined 5,690 Danish Royal Marines comprising non-smokers, individuals with a tobacco consumption of less than 10 gr. per day and those using more than 10 gr. per day. He reported a correlation between tobacco consumption and ulceromembranous gingivitis, between tobacco consumption and deposition of calculus, and between calculus and gingivitis. He produced statistical data which indicated that tobacco has an effect per se on the gingival tissues. Pindborg[34] also reported a similar incidence of gingivitis in cigarette and pipe smokers but a significantly greater deposit of calculus in pipe smokers than in cigarette smokers.

Though there is reliable clinical evidence that occupational hazards and certain habits result in gingival disturbances and periodontal breakdown, there would seem to be little reliable epidemiological data on the effects of occupation or habits on the incidence of periodontal disease.

AGE DISTRIBUTION

In general one can say that the incidence and severity of periodontal disease increases with age. There is also some evidence to support the view that there is a more or less definite pattern of gingivitis prevalence at various age levels. This pattern is not entirely consistent with the first general statement.

Table 2 shows the results of surveys by the same examiner in the same year in four widely separated geographical areas and on four separate racial groups. The comparative figures for the Rochester, N. Y. and Indian groups should be accepted with some reservation because of the comparatively small number in some of the age groups; but despite this qualification a fairly definite pattern can be noted in the four different groups of children. Beginning at age 6 years, the average incidence of gingival disease rises somewhat irregularly to reach a high at about the age of puberty, and then falls quite sharply and consistently in all four groups from 14-18 years of age. Figure 1 shows the mean curve for the four groups, and illustrates the general pattern. Though King did not report figures for the younger age groups he, also, found a lower percentage of subjects affected at age 16-18 than at age 12-14 years, both at Dundee and at Harpenden.

In our results from India it was not always possible to trace the same pattern because of the extreme prevalence and severity of gingival disease in all age groups. Of 1,054 children from 9-17 years of age, examined in one Muslim school, only 6 could be classified as normal, and the average

TABLE II
Percentage of Children with Gingival Disease

Age Group	*Rochester, N. Y. (234 Subjects)*	*Puerto Rico (1488 Subjects)*	*Virgin Islands (823 Subjects)*	*India (249 Subjects)*
6	38	65	42	—
8	76	78	51	74
10	86	76	59	64
12	81	79	64	73
14	75	71	66	82
16	73	65	55	74
18	50	60	54	69

MEAN CURVE SHOWING GINGIVAL DISEASE PATTERN OF CHILDREN 6-18 YEARS OLD

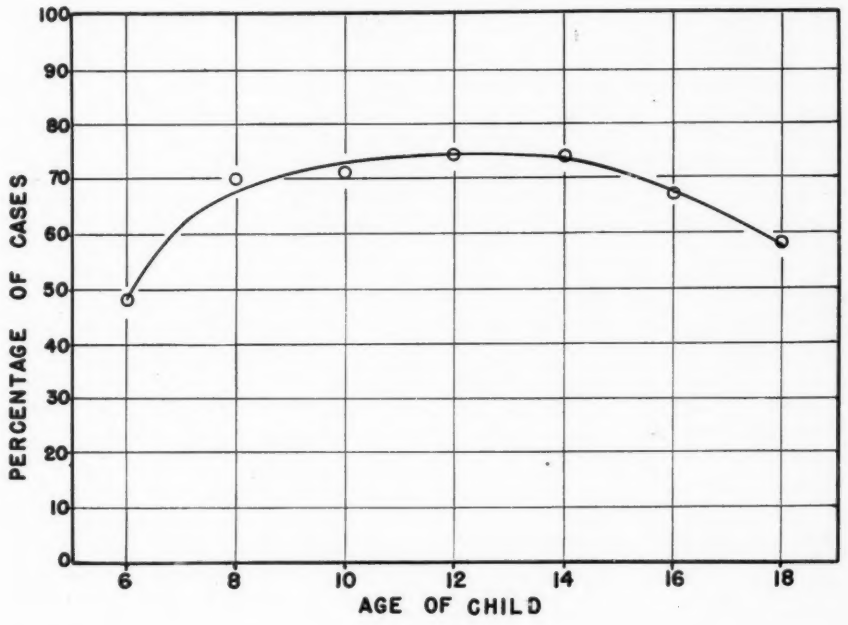


Figure 1.

prevalence ranged from 95-100 per cent; but a somewhat similar pattern was seen in a girls' school attended by children of high socio-economic status where the situation was not entirely obscured by the almost universal prevalence of the disease.[35]

The comparatively low disease incidence among the Virgin Island children, of whom 91 per cent were negro, may possibly be explained on racial grounds.[36]

The percentage prevalence in Table 2 refers to the total gingivitis picture without differentiation of acute and chronic types. Reference may be made to the individual reports for this differentiation.

The groups reported on by Schour and Massler in Italy have not been sufficiently broken down to permit a comparison of their findings with our own in several countries with reference to the pattern of prevalence from 6-18 years of age. They report a steady upward trend from 40 per cent in the 6-10 age group to 98 per cent in the 51-60 year old group.[37]

Following this fluctuating but consistent pattern in gingivitis prevalence in the 6-18 year old group, there would seem to be a steady increase with age towards further gingival disturbance and periodontal breakdown. Only reports based on investigations including roentgenographic examination can be considered reliable. In this connection the work of Miller and Seidler[38] showed that the percentage of subjects with actual bone destruction increased steadily from 9 per cent in the 11-15 year old group to 97 per cent in the 41-45 age group. The higher

age groups showed some slight fluctuations probably due to the greater number of extractions, and of resistant cases not reporting for examinations. Moreover Miller and Seidler's group were hospital patients and may be considered not entirely representative of the population as a whole.

The results of our own investigations in India[23] covering 568 subjects (not hospital patients), though differing in incidence and degree of involvement, showed precisely the same upward trend, from 0 per cent in the 9-12 age group, 8 per cent in the 13-14 age group, 40 per cent in the 15-16 age group to 100 per cent in the higher age groups. The average degree of bone resorption, as seen in the bar diagram (Fig. 2), showed a steady upward trend to age 34 years with some fluctuations thereafter for the same reasons as in the work of Miller and Seidler.

It would therefore appear that there is a somewhat definite pattern in age distribution with regard to both gingivitis and other forms of periodontal disease. The generally high incidence of gingival disease around the age of puberty may be related to hormonal or other systemic factors and the consistent fall in prevalence after the age of 14 may be explained in part on social grounds related to improvement in oral hygiene. Much more epidemiological investigation is required in this country before these and other questions can be satisfactorily answered.

REGIONAL DISTRIBUTION IN THE ORAL CAVITY

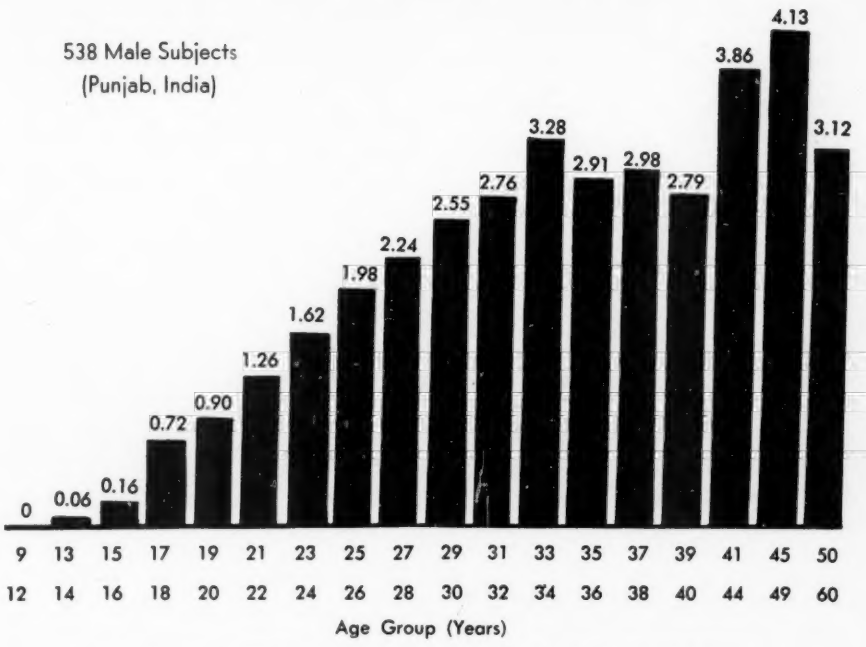
Another epidemiological characteristic of periodontal disease that requires further study is the relative gingival disease and alveoloclastic experience in various regions of the mouth. In our own experience the regions most commonly affected with gingival disease, in descending order of frequency, are: upper labial, lower labial, upper buccal, lower buccal, palatal and lingual, with the lingual tending to replace the palatal region in the older age groups. Our studies emphasized the danger in using one region of the mouth (lower labial or upper labial) as an index of the mouth condition as a whole. A study of the regional distribution of calculus and gingival disease indicates that calculus plays only a negligible role in the age groups examined.[36] With respect to relative alveoloclastic experience in various mouth regions, roentgenographic examination in India[23] of 538 non-hospital male subjects from 9-60 years of age confirmed in part the findings of Miller and Seidler[38] in this country on 500 periodontia clinic patients. An important finding in both investigations was the relatively greater resistance of the alveolar bone in the bicuspid region compared with that in the first and second molar area. This has also been noted by Tenenbaum, et al.[39] In India, however, we found that the lower incisor area was much less resistant than the upper incisor area and had approximately the same degree of resistance as the molar region.

These variations in resistance have been related to eruption time with associated growth changes, and to anatomical features such as formation, position and relation. It is probable that some useful data could be obtained by further studies of this epidemiological characteristic of the disease.

SEX DISTRIBUTION

There has been considerable controversy as to the relative sex distribution of peridontal disease. In Dundee, King reported the percentages of subjects affected by some degree of gum disease were 88.4 and 90 for the boys and girls respectively in one primary school, and 92.5 and 97.5 for boys and girls respectively in another (age 12-14). At 16-18 years of age the percentages were: Secondary schools, boys 80.0, girls 77.5; in factories, boys 92.7, girls 55.6. At Harpenden in England the corresponding figures were 71.4 for boys and 53.8 for girls.[20]

Our own surveys in India, Puerto Rico and the Virgin Islands indicate a consistently lower gingivitis prevalence in female



Bar diagram showing average degree of bone resorption in various age groups

Figure 2.

children. In India the examination of a group of children of high economic level and the same age range (6-11 years) revealed a gingivitis prevalence of 78% for the boys and 71% for the girls.[35] In Puerto Rico examination of 1648 children showed the prevalence of gingival disease in the various age groups averaged from 67-85% in the case of the boys, and 45-74% in the case of the girls. In every age group from 6-18 years the girls showed a significantly lower disease figure.[40] Similarly in the Virgin Islands with a 91% colored population of low economic status the females (54%) showed a significantly lower average prevalence than the males (61%). A total of 823 children was examined. In each age group, from 8-18 years, the females showed a lower degree of prevalence.[36]

In another investigation in India, 760 older patients were examined for evidence of chronic suppurative periodontitis. In all age groups from 21-60 the females showed a lower incidence of disease, but the differences were not particularly significant except in the 21-30 age group in which the percentage prevalence was 71.7 for the males and 52.7 for the females.[26]

In the United States Miller and Seidler[21] reported the need for periodontal treatment in 62% of the males and 56% of the females in 1003 cases roentgenographically analyzed; and a study by the Committee on Economics of the American Dental Association[41] reported a higher average percentage of men requiring periodontal treatment than women in each economic group. Evidence based on our own investigations in several countries and others in the United States would seem to indicate fairly conclusively a lower incidence of gingivitis among female children and adolescents. A comparison of sex incidence in relation to

other forms of periodontal disease and in older age groups, suggested a lower incidence of periodontitis and average degree of bone resorption, but a comparatively higher incidence of periodontosis in female subjects.

Reliable information on this subject and in relation to periodontal changes which occur as the result of endocrine changes during pregnancy and the menstrual period must await the acceptance of periodontal disease as a public health responsibility and the institution of broad-based studies.

SUMMARY AND CONCLUSIONS

Some public health aspects of periodontal disease have been presented and its more important epidemiological characteristics discussed. The reasons for the present lack of reliable information on this aspect of the disease have been considered and the importance of further studies in this field emphasized.

A comparison is made of the results of gingivitis surveys made by the author in several widely separated geographical areas on four distinct racial groups. A definite pattern of gingival disease prevalence has been established. These surveys also show a consistently lower gingivitis prevalence in female children; and emphasize the danger in using one region of the mouth as an index of the gingival condition as a whole.

Results of the limited number of roentgenographic surveys so far conducted to investigate alveoloclastic experience show a considerable measure of agreement with respect to age and sex distribution and regional involvement.

Further investigation is still required on these and other epidemiological characteristics of the disease. With the possibility of improved methods of caries prevention and control and with the expectancy of a relative population increase at the higher age levels, periodontal disease will assume a greater degree of importance. The study of periodontology should be given its rightful place in the dental curriculum and as a preliminary to further fundamental research in this serious public health problem, the various epidemiological aspects of the disease should be thoroughly examined.

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(Continued on page 59)

Studies in the Etiology of Periodontal Disease

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THE etiology of periodontal disease involving alveolar bone loss is still in the main an unsolved problem. The cases chosen in our effort to study the possible etiological factors in this condition were divided into three groups.

The first group, predominantly females, included either young individuals whose radiograms showed signs of severe alveolar bone loss, generally in specific areas, or patients in whose mouths local factors were either absent or not prominent and apparently did not account for the severe loss of alveolar bone.

As a rule the gingivae appeared pink and stippled but there was slight erythema of the interdental papillae in several areas in a majority of the cases. Little or no calculus was present. A diagnostic sign was the migration of the maxillary or mandibular incisors with resulting diastemata.

Radiographic examination revealed loss of alveolar bone that generally conformed to a definite pattern. The bone loss usually extended in an arc from the mesial surfaces of the second molars to the distal surfaces of the second bicuspids. There was also loss of alveolar bone around the incisor teeth that had migrated. In many areas there was a thinning of the alveolar crest and a widening of the periodontal membrane. More alveolar bone was present about the bicuspid teeth in both arches, especially the mandible. These observations were rather constant in this group in individuals under 25 years of age. This condition is probably best characterized as periodontosis.

The second group, also predominantly females, consisted of individuals in whom one or more local factors were more prominent than in the first group. As a rule, the patients in this group were older than those in the first. The gingivae were red and the papillae had a purplish tinge. No stippling was present and the papillae had lost their firmness and appeared glossy and spongy. Many of the teeth showed second and third degree mobility.

Radiographs of this group showed that the pattern of alveolar bone loss described for the first group was absent because of an almost complete breakdown of the alveolar process. However, relatively more alveolar bone remained around the mandibular bicuspid teeth. This condition corresponds very closely with that referred to as periodontitis complex in the terminology adopted by the American Academy of Periodontology.

The third group, approximately equally divided between males and females, consisted of individuals with heavy deposits of calculus. The gingivae were inflamed, hypertrophied, purplish in color and bled easily. In many cases a suppurative exudate oozed from the gingival crevice upon finger pressure. There was no elongation or protrusion of the anterior teeth. As a rule, the teeth showed mobility only in the advanced stages.

Radiographic examination revealed a horizontal loss of alveolar bone which was severe in a majority of the cases. This condition is perhaps best characterized as an advanced marginal periodontitis.

The investigation included a comprehensive dental examination, blood chemistry, blood count, basal metabolism, dietary record, Kline test, physical examination, vaginal smears, urinary determinations of: neutral 17-ketosteroids, estrogen, thiamin, riboflavin, and methylnicotinamide; and salivary determination of calcium, phosphorus, pH and protein.

There were 51 cases in the control group (individuals free from periodontal disease)

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with a mean age of 33.5 years, 77 cases in the periodontosis group, mean age 27.3, 59 cases in the periodontitis complex group, mean age 39.1, and 39 cases in the advanced marginal periodontitis group, mean age 48.8.

Blood constituents for which differences were found between the control and each of the periodontal groups include vitamin C, glucose, globulin, albumin, calcium ion, alkaline phosphatase, red blood cell sedimentation rate, red blood count, white blood count and neutrophiles. The deviations from the normal ranges were, in general, small. No appreciable differences were found between the control and experimental groups for total calcium, inorganic phosphate, sodium, chloride, total protein, total cholesterol, free cholesterol, non-protein nitrogen, urea, uric acid, carbon dioxide capacity, hemoglobin, vitamin A and carotene, although deviations from the normal ranges were obtained for both groups. Likewise, no difference was found between the two groups for the basal metabolic rate. The Kline test was negative in all cases.

The dietary study consisted of a record of the food eaten during a one week period. From the information thus obtained, the intake of some of the essential food constituents was calculated. On the basis of the recommended daily allowances of the Food and Nutrition Board, low caloric intakes were found in 45% of the control group and in 48% of the periodontosis groups. Results of a similar nature were found for protein, calcium, phosphorus, iron, thiamin, riboflavin and niacin. In each of these instances there was no fundamental difference between the two groups. It is clear, therefore, that a large number of cases in the control and periodontosis groups do not measure up to the standards set by the Food and Nutrition Board for the food essentials listed. However, the significance of this finding is greatly reduced inasmuch as the recommended dietary allowances are admittedly higher than average requirements, and the deviations from these allowances were, in general, small. The results for vitamin A and C are in striking contrast to the other dietary constituents. Our calculations indicated that with few exceptions adequate amounts of these two vitamins were ingested by both groups.

The possibility of a subclinical deficiency of thiamin, riboflavin and nicotinic acid was studied also by urinary determinations. These tests are based on the hypothesis that if thiamin, riboflavin and N^1-methylnicotinamide are present in urine collected during an arbitrary period, as for example, during the 13th hour after a meal, it indicates that the body has a surplus available for excretion. On the other hand, if excretion of these substances during the specific period falls to zero, it indicates that no surplus is available for excretion, and that such an individual is potentially deficient in these vitamins.

Our analyses indicated that these vitamins were present in all of the one-hour excretion samples. Therefore, these tests did not reveal a subclinical deficiency of thiamin, riboflavin or nicotinic acid in any of the subjects. Furthermore, when the approximate amounts of these substances in the control and experimental groups were compared, no differences were noted between the group.

ENDOCRINE ACTIVITY

Since endocrine activity may be involved as an etiological factor in periodontosis, one of the studies undertaken was the determination of the neutral 17-ketosteroids in urine. These substances represent excretion products of some of the steroids of the adrenal cortex, while some of them arise in part from the male gonads. Therefore the determination of the 17-ketosteroid output is essentially an index of adrenocortical and male gonadal activity. In the present report, results are presented for only the females in our series since too few males have thus far been studied. There were 17 cases in the control group for which a mean value of 11 mg. per 24 hours was obtained.

The periodontosis group consisted of 25 cases with a mean of 9.2. The difference between the means divided by the standard error of the difference is 1.8, which means that the odds that the difference is not due to chance are 13 to 1, which is somewhat below the borderline of significance. It is of interest to note that 30% of the cases in the control group were below 9.2 as compared with 60% in the periodontosis group.

Since the great majority of periodontosis cases were females, tests were carried out which might throw light on ovarian function. This was done by determining the estrogen content in urine and by making vaginal smears.

For the estrogen determination, 24 hour urine specimens were collected generally on the 7th day of the menstrual cycle, and on the day on which there was a marked rise in temperature. In the usual 28-day cycle this generally occurs at some time between the 11th and 15th day of the cycle, at the time of ovulation, and the increase in temperature is about one degree Fahrenheit. If the temperature did not rise by the 15th day, a 24 hour specimen was collected on that day or the following day.

Results have been obtained thus far for 13 cases. The range during a normal cycle is approximately from 15 to 50 gammas per day. The estrogen output decreases during menstruation. There is a peak excretion at mid-interval and another just preceding menses. With the exception of one case, all values were within the normal range.

Vaginal smears were made on alternate days during a complete cycle beginning with the first day. The smears were made by the patients after detailed demonstration and instruction. Eleven cases have been studied thus far. With one exception the cycles were ovulatory according to the indices employed, that is, the degree of cornification, evolution of cell type, and temperature. In these cases the quality of the follicular phase varied from poor to excellent. It was poor in one case, fair in three, fair to good in two, good in two, and excellent in two. However, luteal phases appeared to be normal in all of these cases. In the one instance that was anovulatory the estrogen output was low, and only at the very end of the period studied, a small amount of cornification appeared.

Physical examinations and case histories in 44 of the periodontosis patients gave essentially negative findings in 17 of them. In the remainder of the group, a number of apparently unrelated conditions were revealed. These included the following:

Systolic murmur (or mitral stenosis) in 6 cases
Diabetes in 3 cases
Hay fever in 3 cases
Mild hyperthyroidism in 2 cases
Intermittent dyspepsia in 2 cases

and the following, each in one case:

Enlarged liver and tender spleen
Amoebic colitis
Recurring peritoneal abscesses
Hysterectomy because of fibroid tumor
Recurring maxillary sinusitis
Hodgkin's disease
Cervical lymph node tuberculosis
History of syphilis and gonorrhea
Seborrheic dermatitis
Cystocele and palpable right kidney
Thickening and roughening of the skin and moderate hirsutism.

It is clear that no consistent pattern has been found.

In view of the importance of dental calculus as an etiological factor in periodontal disease, salivary analyses were made in patients with and without calculus to determine the relationship of the composition of saliva to the tendency to form these deposits. Inasmuch as the greater part of the mineral matter in calculus consists of calcium phosphate, it was felt that the concentration of calcium and phosphate in saliva may be associated with the tendency to form these deposits. Furthermore, precipitation of calcium phosphate depends upon the type of phosphate ion present, which in turn is dependent on the hydrogen-ion concentration of the medium. Hence, a relationship may exist between pH and calculus formation. Also, the concentration of protein may be important

inasmuch as the precipitation of calcium salts is markedly reduced by the addition of soluble protein to a solution. In view of these considerations, calcium, phosphorus, pH and protein determinations were made in a group of persons who tend to form heavy deposits of supragingival calculus, and in a group that showed very little or no tendency to form such deposits.

The "calculus group" included individuals who had heavy deposits on the lingual surfaces of the lower anterior teeth. Most subjects, however, had heavy deposits not only on these surfaces, but also on the buccal surfaces of the upper molars, or throughout the mouth. The calculus-free group consisted of individuals who were free from supragingival calculus, although they had not had dental prophylactic treatment for at least one year. The age range of the subjects studied was between 20 and 60 years.

The most striking difference between the calculus-free and calculus groups was found in the calcium content of both unstimulated and stimulated saliva. The mean values for calcium were lower in the calculus-free than in the calculus group, the difference being statistically significant. The mean values for inorganic phosphate were likewise lower in the calculus-free group for both types of saliva, the difference between the two groups being statistically valid in the case of stimulated saliva. For unstimulated saliva, the difference between the means is on the borderline of significance. No significant differences between the groups were found for protein in either unstimulated or stimulated saliva, or for pH in the former. However, the difference between the groups for the pH of stimulated saliva may be regarded as significant.

These findings indicate that the average concentrations of total calcium and inorganic phosphate in both stimulated and unstimulated saliva, and the average pH of stimulated saliva, of persons who form calculus, are relatively such as to favor the precipitation of the calcium phosphate found in the deposit.

SUMMARY

The fact that in the blood analyses more deviations from the normal were found in the periodontosis and periodontitis groups than in the control group suggests that one or more systemic conditions are associated with these diseases in many of the subjects studied. No clear distinction in blood chemistry was found between the periodontosis and periodontitis groups. From the available data, it does not appear that a common systemic condition operates as a modifying or predisposing factor in these conditions. It is quite possible that in some instances, as in the cases of phosphatase, erythrocyte sedimentation rate and leukocyte count that the relation may be one of effect rather than of cause.

The physical examinations did not reveal a common systemic condition responsible for the disease.

The dietary study did not show any difference between the control and periodontosis groups in the intake of certain dietary essentials. The fact that plasma vitamin C was low in a large percentage of the periodontosis cases in spite of an adequate intake of this vitamin may mean that there was some interference in the utilization of this vitamin.

A low urinary estrogen output was found in one of 13 periodontosis cases. On the basis of a study of vaginal smears, subnormal follicular phases were found in 5 of eleven cases.

Although a difference was found between the control and periodontosis groups in the ketosteroid content of urine, it is as yet uncertain whether adrenocortical dysfunction is related to periodontosis.

Salivary analyses indicate that the calcium and phosphorus content of saliva is related to the tendency to form calculus.

Calculus Formation*

By HAROLD C. HODGE, *Rochester, New York*

FROM chemical analyses, the mineral part of calculus has been shown to be very much like the mineral part of tooth and bone substance. The space lattice of this calcium phosphate is hydroxylapatite.

From the data of Ericsson (Acta Odont. Scand., Vol. 8, Suppl. 3, 1949), the solubility of powdered enamel in saliva at various pHs has been shown to follow the same straight line as does the solubility of a synthetic hydroxylapatite in saliva at comparable pHs. No such solubility data are available for calculus; it is assumed that the solubility of calculus in saliva would follow the same straight line as had been found for enamel and for hydroxylapatite.

It is of interest to ask whether saliva as secreted is saturated, undersaturated or supersaturated with respect to enamel (to hydroxylapatite and to calculus). The data of Schmidt-Nielsen (Acta Odont. Scand., Vol. 7, Suppl. 2, 1946) on the composition of human saliva show that mandibular saliva is supersaturated and paratoid saliva is undersaturated with respect to enamel. The saliva of calculus-free and calculus-forming patients analyzed by Tenenbaum and Karshan (J.A.D.A., 24:1255, 1937) was supersaturated in both instances. However, the calculus-forming group was more supersaturated. Saliva should be at least saturated with respect to enamel to maintain integrity of the mineral phase.

The loss of carbon dioxide may be important in calculus formation. Wah Leung in his thesis (1950) analyzed saliva before and after equilibration with various air concentrations of CO_2. As secreted, the saliva was supersaturated; with CO_2 loss, calcium and phosphorus were lost.

*Abstract of presentation. For a more detailed report see article by Drs. Hodge and Leung in the Jour. of Perio. 21:211 (1950).

Summary of Systemic Relations in Periodontal Disease

By DONALD A. KERR, *Ann Arbor, Michigan*

DISEASE processes which involve the body as a whole are referred to as general or systemic diseases and are believed to play a part in the production of periodontal disease. General disease, as an etiological factor in periodontal disease, has been assigned varying degrees of significance by different authors. The association between systemic disease and periodontal disease has been more theoretical and ethereal than actual and scientific. Positive systemic factors cannot be demonstrated in most cases which suggest a systemic etiology. However, it has been observed that periodontal disease in a severe form is often associated with certain systemic diseases. It is this state of finding severe periodontal disease associated with systemic disease that has suggested the possible cause and effect relationship, rather than the opposite situation of observing the severe periodontal disease and demonstrating the systemic etiological factor. Therefore, this afternoon it will be my purpose here to enumerate a number of systemic conditions which are found in association with periodontal disease and try to point out how they may be operative in affecting the periodontal disease.

My first remarks will concern periodontitis and later remarks will be directed toward periodontosis. When periodontosis has become periodontitis complex, it can then be considered in the remarks on perio-

dontitis. In periodontitis I will divide the systemic factors into three broad groups.

GROUP I: TOXIC PROCESSES

In this group one should consider the toxicoses produced by heavy metals such as bismuth, mercury and lead. In mercury and bismuth toxicity there is a direct effect on the periodontal tissues, resulting in necrosis or marked reduction in tissue vitality, which accentuates the importance of the local factors which may be present. The severity of the periodontal disease is dependent upon the potency of these two related factors.

Auto-intoxication has been suggested by many as a possible systemic etiological factor. Auto-intoxication indicates the formation of some substance in one part of the body which is toxic to some other part or the body as a whole. Since there is not uniform agreement that such a condition as auto-intoxication exists, it is then not reasonable to classify it as a systemic etiological factor.

GROUP II: ENDOCRINE

Endocrinopathies, especially diabetes mellitus, have been given considerable importance as a factor in the production of periodontal disease. Both hyper- and hypothyroidism have also been related to periodontal disease in an important role. The other endocrine disturbances have been associated to a lesser degree. It has been shown by Glickman and others that diabetes is never the initiating factor in periodontal disease, but its presence will markedly alter the course of periodontal disease. It seems entirely reasonable that all endocrinopathies, especially hypo-insulinism, hypothyroidism and hyperthyroidism should alter the course of periodontal disease. They are diseases which affect the metabolism of the entire organism and therefore alter the resistance and reparative ability of tissues. When periodontal changes are present due to local factors, the degree and rapidity of destruction is controlled by the ability of the tissue to resist damage and repair the destruction produced by the local factors. If, upon the effect of the local factors is superimposed an alteration in cell metabolism, it will result in lowered resistance and reparative function and, therefore, a more rapid progress of the disease. It is the alteration of the tissue metabolism which acts as a contributory factor. This is important in diabetes mellitus and thyroidism; they alone do not produce periodontal disease but do affect greatly its course.

GROUP III: DEBILITATING DISEASES

In this group are the deficiency diseases, especially avitaminosis C. In all deficiency states there is an alteration of cellular metabolism which affects the resistance and reparative ability of tissue and will therefore alter the progress of disease. This is especially true in avitaminosis C because it affects directly the metabolism of connective tissue. Evidence indicates that these conditions are never etiological factors, but are contributory and alter the course of disease after it has been initiated by causative factors.

In this group we may also consider such disease processes as allergy, nephritis, tuberculosis, and even psychosomatic processes. All may produce some alteration in the general metabolism, which affects indirectly the periodontal disease.

Another condition to be considered in this group is senility. In considering the age incidence of periodontal disease, it is clearly evident that it is primarily a disease of middle age and the incidence and severity of the disease increases with age. This is reasonable and should be anticipated, because with increase in age the processes of anabolism and catabolism become more nearly equal. In the young the stimulus of growth is potent and the cells have a great proliferative ability. With increase in age the proliferative or reparative ability of cells decreases while the destructive processes are intensified. The result is inability of the individual to compensate for damage produced by local factors and the periodontal

disease is thus progressive.

PERIODONTOSIS

In periodontosis a general disease state may be of much greater significance. Periodontosis is characterized by a degenerative process in the connective tissue of the periodontal membrane. The several disease processes, such as periarteritis nodosa, lupus erythematosis, dermatomyositis, etc., are classified as collagen diseases and are characterized by degeneration of collagen fibers. Because of the close similarity of the pathological changes in the collagen diseases and periodontosis, it may be that periodontosis is a manifestation of collagen disease. This is hypothetical, but the similarity of pathological changes is striking enough to consider it a strong possibility.

In summary it may then be suggested that systemic disease is not an etiological factor in periodontitis except in cases of severe acute mercury and bismuth poisoning. In all other instances systemic disease is a contributory factor which may alter the course of periodontal disease after it has been initiated by other factors. Periodontosis, however, may actually be a manifestation of systemic disease. It may be a manifestation of collagen disease which presents a specific pathological and clinical change not seen in periodontitis.

Changes in Bone and Cementum in Periodontal Disease

By JOSEPH L. BERNIER, D.D.S., M.S.,* *Washington, D. C.*

ALTHOUGH certain of the changes in alveolar bone which occur in periodontal disease are presented in unusual patterns, this is, in part, the result of local functional and environmental factors and therefore demonstrates the physiological maneuverability of bone as a tissue. It is well established that many factors and states are important in the etiology and pathogenesis of periodontal disease and that of these, infection and its attending inflammatory changes as they affect the periodontium, are probably the least understood. This is so not only because the inflammation in itself is complicated, but because the reaction of calcified tissues to this process is extremely variable. This discussion inquires into the nature of the influence of inflammation on the calcified tissues of the periodontium and does not offer any solution as to the role it may play as a specific etiologic factor.

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It seems important to establish an initial premise that newer studies into the physiology and histochemistry of calcified tissues, particularly bone, suggest a process of greater simplicity than heretofore believed. This may add much information to that now at hand and perhaps suggest new theorems as to the influence of infection and inflammation in periodontal disease. The selection of this reaction from the many others that operate in periodontitis as a likely port of entry into a study of the changes in the calcified tissues of the periodontium stems from a firm belief, as Rosebury[1] has stated that "periodontal disease means to me any inflammatory process of the gingival and periodontal tissues and its precursory states except those of a specific infective character. Tumors and other conditions of a specific nature are thus eliminated, leaving three clinical conditions—gingivitis, Vincent's infection, and periodontitis, all of which are fundamentally similar and which overlap clinically and

histologically, making separation difficult."

There are obvious practical advantages in assuming that the clinically healthy mouth is never free of inflammation, a point which appears to have been well substantiated. In this connection recent studies at the Armed Forces Institute of Pathology involving the histologic analysis of over 7000 gingival specimens, most of which were taken from clinically normal interdental areas, suggest the presence of early inflammatory changes, as measured in terms of edema and cell proliferation, in a high percentage of all specimens examined. Study of additional material continues to sustain this observation. Final results are not yet completely tabulated so that this is not offered as conclusive evidence. This and other observations, however, tend to indicate the presence of infection and inflammation in periodontal disease in its sub-clinical states, a point which may be of great importance in evaluating clinical periodontitis.

It is only necessary to mention the importance of the endocrine glands and nutrition in the formation and physiologic maintenance of calcified tissues. The reports of Becks et al.,[2] Evans,[3] Erdheim,[4] Smith and McLean,[5] Silberberg,[6] and Ellsworth,[7] are particularly complete on these points. It must be stated, however, that their influence when associated with infection and inflammation as in periodontal disease is of great significance even though it is not well understood.

From a comparison of the refractive indices and x-ray diffraction pattern of bone, dentin, cementum, enamel, and certain crystalline minerals composed mainly of calcium and phosphorus, Taylor and Sheard[8] have concluded that these substances are similar in that they contain minute crystals of podalite of the apatite series. Other reports[9,10] on the protein chemistry and growth patterns of these tissues suggest additional points of similarity. Of these calcified tissues, bone and cementum seem most closely allied so that observations on the reaction of bone and cementum to infection and inflammation can, in many instances, be made to include both. It should be remembered, however, that although their similarity is physiologically fundamental, there can be no doubt as to the many points of difference in the peripheral reaction of these tissues, particularly as related to the influence of functional environment.

THE PROCESS OF CALCIFICATION

Since it is well known that resorption of bone occurs in periodontal disease, it is well to consider briefly some of the theories of calcification in that demineralization, in a manner not yet fully understood, is part of the resorptive process. Bone matrix is chiefly a protein tissue consisting of ossein (osseocollagen) with small amounts of osseomucoid and albuminoid. This matrix material is arranged in a fibrillar pattern, the fibers being drawn into small bundles and united with an intercementing substance. The intercementing substance contains many of the inorganic salts and is afibrillar. Halisteresis—the removal of products of calcification from living bone—probably does not occur as such. However, in the resorption of bone, when the osteoclasts, by proteolytic action remove the organic constituents of bone, the calcium salts are liberated, perhaps to be carried away by macrophages.

A number of theories have been advanced to explain the process of calcification, all of which are incomplete in one or more details. One theory suggests that calcium is adsorbed by a protein constituent of cartilage for which it has a special affinity, the calcium combining the phosphorus to form tricalcium phosphate. Another theory suggests that the precipitation of calcium phosphate from a saturated solution in a solid form is dependent upon the product of the concentrations of calcium and phosphate ions in the solution and not upon the total quantities of these substances present. Further, that protein in the solution reduces the degree of ionization and that carbon dioxide in the body fluid decreases its

ability to hold calcium salts in solution. A reduction of either of these two factors would then seem to favor the deposition of calcium salts. Watt,[11] has suggested that calcification is not a simple precipitation of calcium phosphate from solution but is dependent upon the active secretion of bone salts by the osteoblasts formed intercellulary from inorganic constituents of the blood. Robison,[12] believes that calcification is dependent upon the action of the enzyme phosphatase which is contained in certain elements of bone and is capable of hydrolyzing phosphoric esters. He believes it to be the product of the osteoblasts, proliferating cartilage cells and cells of the inner layer of the periosteum. There is much evidence to support this contention. The full significance of the enzyme phosphatase in bone formation and in calcification is not as yet understood, however, there is strong evidence to suggest that the osteoblast is rich in alkaline phosphatase and the osteoclast in acid phosphatase. This, according to some observers is one of the fundamental differences between these two cells and may be largely responsible for determining which function either will perform. Further, it has been suggested that the osteoblast and osteoclast may be the same cell, either forming or destroying bone as its phosphatase fraction is altered.

Time does not permit a detailed discussion of the inflammatory process but, since it is well known that alterations in pH, alkaline reserve, CO_2 levels, protein and carbohydrate metabolism are produced it is easy to suppose that inflammation might affect the completeness or, for that matter, the occurrence of calcification.

THE SPECIFIC INFLUENCE OF INFECTION AND INFLAMMATION ON BONE

In gingivitis the distribution of the inflammatory exudate is largely confined to the area above the transeptal fibers, the alveolar bone becoming involved only as the reaction spreads. Of particular interest is the character of the cellular exudate which tends, even at this stage, to demonstrate an increasing number of plasma cells. Although the exact role of this cell is not well understood, it is, together with the lymphocyte, becoming more important as its function in the immunity mechanism of the inflammatory response becomes clarified. It has been demonstrated that both of these cells are important in the elaboration of immune globulins of various types, not only at the actual site of the injury and reaction, but also at distant parts of the body where lymphocytes and their derivatives are formed, such as lymph nodes. It is possible that the function of the lymphocytes and their derivatives, the plasma cells, although mechanically not impressive, may turn out to be the most effective element in the control and destruction of invading organisms. This is suggested by their appearance and quantity when a sustaining influence is present. If this is true it may be possible to state that in periodontitis, where these cells are prominent, the inflammatory process is initiated and sustained by extra-ordinary influences requiring the full mobilization of these cells in quantity. These unusual factors should, in my opinion, be considered as associated with function as related to mastication since a majority of patients with fulminant periodontal disease are free of serious systemic disease. In this connection one must admit the difficulty in explaining the reaction of the periodontal membrane in the face of such influences since both proliferation toward repair and retrogressive changes toward necrosis are noted. It seems likely that this contradiction of events should be considered as being associated with disturbances in cell chemistry and not with any alteration in cell potential. This, in my opinion, is so since in inflammation, cell potential is not in conflict with the factors responsible for either benign or malignant neoplasia. Even though there is some reason to believe that these influences may be related directly to cell metabolism, they appear to be controlled in their intensity by inherent genetic factors.

It is difficult to avoid references to the classification of periodontal disease since it is from such academic efforts that one must

select terms. The so-called hypertrophic and hyperplastic changes in the gingivae associated with gingivitis, need not, except in a clinical sense, be given special consideration in periodontal disease since again environment and function seem to be important in their development, the basic reaction remaining unchanged. One might liken these changes to the "laryngeal nodes" occurring on the vocal cords and in the aryepiglottic fold when trauma operates to excess. Because of suggested specificity in etiology and difference in tissue change, the gingival changes in pregnancy do however deserve special mention so that this reaction need not be contained in the "waste basket" of nonspecific clinical reactions. This is, in my opinion, equally true of the so-called "fibromatosis gingivae" which I prefer to consider as a benign neoplastic response. One is indeed hard pressed to explain what to him connotes morphologic benign neoplasia, however, the fact that the usual forms of clinical treatments fail to result in a diminution in cell proliferation in this condition suggests aberration in growth qualities of the cells beyond that seen in cell proliferation associated with inflammation which can, in many instances, be caused to regress.

HISTOLOGIC CHANGES

When in marginal periodontitis the alveolar crest becomes involved in the inflammatory process resorption becomes evident. This represents an extension of the inflammation with a resultant alteration in the osteogenic tissues adjacent to the alveolar process which apparently emphasizes the role of the osteoclast. Preceding this, however, it is possible to detect changes in the marrow substance of the alveolar bone which initially are represented by dilatation of the blood vessels and later by fibrosis. These changes occur before the substance of the periodontal membrane is affected in terms of loss of tone, disintegration of fibers, and resorption on the periosteal side of the alveolar bone. Extension of this reaction apically brings into play further modifications in function and environment which may tend to delay the further involvement of bone or in direct reverse, to accentuate its destruction. This is so since inflammation, under certain conditions, tends to be self-limiting. For the most part, however, the destruction continues, perhaps intermittently, to involve more of the periodontium. It is difficult to understand, therefore, why emphasis has been placed on certain clinical phases of this extension of the inflammation, even to the point of considering certain of these phases as specific clinical and histopathologic entities. Glickman,[13] has recently stated "on the basis of microscopic appearance, bifurcation involvement presents no histologic features which differentiate it from pocket formation in the absence of bifurcation involvement. There is, therefore, no justification for the impression that bifurcation involvement is a specific pathologic entity." Further—"the same principles which govern the treatment of periodontoclasia in general, are applicable to the management of teeth with bifurcation involvement." From this one may conclude that there has been a tendency to complicate the clinical reactions in periodontitis as indicated by the necessity to deny the existence of such specific conditions.

The physiologic growth of alveolar bone involves both osteogenesis and osteoclasis and is continuous in varying degrees throughout life. Deposition of alveolar bone is not uniform around the entire wall of the alveolus, being accentuated near the apex and, in general, on the distal surface. This accounts for the slight, though continued, movement of the tooth mesially, with resulting loss of bone on the mesial surface. Such a delicate balance is obviously best maintained in a healthy state free from the effects of inflammation, traumatic occlusion and systemic disease. The cementum likewise is deposited continuously and although its deposition is greatest near the apex, it is formed over the entire tooth root. Its accentuated formation near the apex undoubtedly compensates, in part, for the loss of coronal tooth substance re-

sulting from attrition and abrasion. Cementum is deposited as a cementoid tissue which, like osteoid tissue, is markedly resistant to resorption. Thus the tooth is protected against excessive resorption wherever the cementoid tissue is present in sufficient quantity to act as a barrier maintaining the anatomical continuity of the tooth even in fulminant inflammation when active resorption is present. Hyperplastic reactions of the cementum and its antecedent tissue, the periodontal membrane, cannot easily be separated even though the formation of cementicles free in the periodontal space is sometimes considered as independent from hypercementosis and the formation of cemental spurs. There can be little doubt that both infection and trauma play an important role in hypercementosis, while, on the other hand, functional demands seem to be important in the formation of cemental spurs. Cementum, like bone, is influenced by the effects of inflammation in both directions. It may be stimulated to hyperactivity or repressed when associated with partial or complete necrosis. Superimposed upon these two extremes is the constant alteration in masticatory function which, in turn, is directly referable to these inflammatory states. Thus, we have constantly changing conditions which vary not only in different individuals, but in the same mouth so that again—the importance of function, both physiologic and pathologic—is emphasized.

SUMMARY

An inquiry, such as this, into the effect of infection and inflammation on the tissues of the periodontium serves only to emphasize the complexity of periodontal disease by demonstrating the many factors involved. There seems to be little doubt that infection and inflammation are of prime importance and that there is a lack of information as to their true nature. This, coupled with the inability to completely explain the reaction of soft and calcified tissues to these processes makes an appraisal of the tissue changes in this disease most difficult. To this we must add the important factor of altered masticatory function and, in many instances, systemic dysfunction.

Pathologists have observed and recorded most of the tissue reactions seen in periodontal disease using the timeworn and perhaps now somewhat obsolete conventional microscope. This has been recognized for some time so that we have been guilty of merely re-evaluating what we have seen time and time again, in the hope that some new pronouncement could be made. We now are passing through a period during which the newer methods of microscopy and histochemical analysis are being perfected so that the deficiencies of past technics becomes more obvious from day to day. There has been a tendency to complicate the histologic changes in periodontal disease by separating the fundamental process of inflammation into so many clinical compartments that the inability of therapy to be equally compartmentalized has nullified our efforts.

Studies into the histopathology of periodontitis have been of extreme importance since they have resulted in an accurate appraisal of the progressive changes in this disease. It seems realistic, however, to recognize the limitations of present methods and to actively engage in the perfection of those newer ones which appear to have promise.

In this discussion I have purposely avoided detailed references to the histologic alterations in periodontal diseases, particularly periodontosis, since, as stated before, these changes have been adequately described. It is obvious, however, that a consideration of the histologic and chemical reactions of bone and cementum in periodontitis involves a complete and detailed analysis of the many factors mentioned in this paper, particularly the mechanism of calcification and the role of the plasma cell in inflammation. And since these problems are awaiting additional information only a recognition of their importance is possible at this time.

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Orthodontics and Periodontal Disease

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INTRODUCTION

THE growth in stature of the science of periodontology should be attributed in large measure to the increased emphasis which has been placed on the biological reactions of the supporting tissues of the teeth. Specifically, these reactions have centered around the anatomy, physiology and pathology of these admittedly unique structures, inclusively referred to as 'the periodontium.' It is in the field of histopathology that dental researchers have accomplished their greatest work, and have given to dentistry the large body of knowledge which has been so effectively used in placing all branches of dental science on a thoroughly bio-scientific foundation. Periodontology has taken particular advantage of these contributions by the histopathologists, and as a result, has gone far in attempting to understand the biological background of a disease condition which has plagued mankind for centuries, and had gained for itself a most persistent reputation of incurability.

When similarly intrinsic contributions have been made by dental researchers who will attempt to acquire further information about periodontal histophysiology, and more significantly, accept the challenges of periodontal histochemistry, then it is to be expected that there will be a much less disturbed understanding of those degenerative histometamorphoses which still perplex the periodontist, who, in sheer defense, must continue to refer to "hereditary allergies" and "medicinal idiosyncrasies" and "idiopathic etiopathologies" . . ., terminologies which are delightfully euphonious and eso-

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teric, but are nonetheless inexact and devoid of specificity.

There are some extensions of this state of affairs into other branches of dentistry, just as there are in the cases of many specialties of medicine, so that the periodontist need not feel any more sensitive or apologetic than should any honest empiricist who becomes convinced of the benefits of scientific objectivity and makes the appropriate adjustments.

The utilization of mechanical appliances to assist in the solution of oral problems may be regarded as the general function of orthodontics. More specifically, orthodontics may be defined as that branch of dentistry which has for its objective the prevention and correction of oral anomalies and the harmonizing of the dental structures involved, so that the dental mechanism will function in a normal way. It utilizes an indispensable understanding of growth and developmental changes, and aims at the achievement of proper anatomical and functional relationships of the dental mechanisms.

When there are recognized the innumerable relationships which exist between the physiology of growth and development in oral structures, and the pathological abnormalities produced by and accruing from the inharmony of the dental mechanism of which the periodontium is a most important entity, then it does appear as if there should emerge more definite and concrete contributions which the specialty of orthodontics can continue to make to the science of periodontology.

A. ORTHODONTICS AND EXPERIMENTAL PATHOLOGY IN PERIODONTOLOGY RESEARCH

A review of the literature reveals a tremendous amount of original study which has been and is being accomplished in the area of experimental pathology. These experiments have in general been directed towards finding out the effects produced in the normal periodontal tissues as a result of various orthodontic, traumatic, nutritional and other influences.

1. ORTHODONTICS RESEARCH

In orthodontics research, several studies have been effected with the aim of obtaining a better knowledge of the results of tooth movement. The experiments of Sandstedt and Oppenheim were among the first to point out that bone resorption and rebuilding occur on each side of a moving tooth, but in a different order. Johnson, Appleton and Rittershofer used young monkeys and located the fulcrum point of the tooth as being somewhere between the apex and the middle of the tooth root. Schwartz experimented on dogs and determined the amount of force required to crush the periodontal membrane between the root and the alveolar wall. Marshall used strong forces in experimenting in tooth movement in rhesus monkeys and produced fractures of the alveolus and extensive dentinal and cemental resorption. Gottlieb, Orban, Weinmann and others made intensive studies and observed the tissue reactions to orthodontic forces used in the movement of human teeth. Their studies revealed injuries similar to those produced in animal experimentation. Urban, Beisler and Skillen concluded that damage to the periodontal membrane was due to rapid separation and the amount of injury was in proportion to the amount of force applied.

Necessarily, there has been a complementary overlapping in areas of researches carried out in orthodontics and periodontology, so that it has been difficult if not impossible to draw a clear line of demarcation between the contributions to dental knowledge which have been made specifically by orthodontists and specifically by periodontists. Actually such a clear cut line of demarcation is unimportant, although academically it may be a source of stimulating argument.

2. PERIODONTOLOGY RESEARCH

Box, one of the outstanding contributors

to periodontology research, experimented on sheep to observe the effects of excessive occlusal stresses. Coolidge has indicated that abnormal and functional stresses result in pathologic changes in the bone, periodontal membrane and root surfaces. Coolidge also indicated that in human jaws, the pathologic changes produced were similar to those produced with orthodontic appliances in experiments on the teeth of animals in which an excessive amount of force was applied. Kronfeld, and later Goldman, pointed out the limited value of the results obtained in animals because of the possible differences in tissue reaction between man and animals. Kronfeld stated also that in individuals with high resistances, even a great amount of occlusal trauma does not produce resorption of the root, while in patients susceptible to root resorption, normal mastication or careful orthodontic treatment may occasionally produce progressive root resorption leading finally to complete destruction of the root. Stillman and McCall point out that in one case a tooth subjected to "plus-occlusion" may become strengthened and in another case weakened, depending upon the ability of the supporting tissues to adjust themselves to altered occlusal conditions. There are, of course, many other outstanding contributions of orthodontics and periodontology, mention of which space does not permit.

The fact does remain that despite these basic and significant studies, there is still a need for further work to obtain a more accurate knowledge of the noxious, and more important, the beneficial effects of various orthodontic, traumatic, nutritional and other influences, upon the periodontium when the latter is affected by periodontal disease of varying degrees of severity. Mershon stimulates a very sobering thought when he points out his fears about the high percentage of cases in which orthodontic treatment has done harm in contrast to the undetermined number of cases in which there have been material and lasting improvements in orthodontically treated cases.

The point is more clearly focused when consideration is given to the fact that orthodontists insist on gingival and periodontal conditions being treated successfully prior to orthodontic procedures. In considering the steps to be taken in orthodontic treatment of cases with existing oral pathological lesions, Moyers thinks that first the intraoral roentgenograms should be examined for regions of bone resorption and radiolucency, then proper therapeutic procedures should be instituted. Finally a sufficient length of time should elapse in order to insure a normal picture of the bone and mucosa *before* instituting orthodontic therapy. When attention is given to the necessity of utilizing orthodontic intervention in treating periodontal disease it is clearly seen that there is need for more specific information in this area.

B. ORTHODONTICS AND PERIODONTAL DISEASE ETIOLOGY

1. INCORRECT ORTHODONTIC PROCEDURE

The correct form and position of the teeth will protect the gingivae from the traumatic irritations of mastication, will help to keep the cervical areas clean, and will aid in the maintenance of proper occlusion and sound alveolar support. In a significantly large number of cases, periodontal disease may be initiated by an irregular alignment of the teeth. The interdental tissues are squeezed by the crowding of the teeth, and food and oral debris are retained in the proximal spaces which are difficult to keep clean. The blood circulation of these tissues becomes impaired and there is likely to occur a gingival inflammation with or without hyperplasia. Other malocclusions such as excessive overbite, mandibular protrusion and improper faciolingual position may produce similar inflammatory conditions.

Gingivitis may not be a serious factor in producing or affecting dental malalignment. On the other hand, periodontitis may produce changes in the periodontal structures which may have potential orthodontic

manifestations in tension, tilting, drifting, loosening and even tooth loss. As in gingivitis, these manifestations may be the cause or effect of disharmony of the masticatory apparatus, and will be alleviated only when there is correction of the orthodontic conditions.

Whenever possible, the realignment of malaligned teeth should be performed by the orthodontist. When this is done without due regard for basic orthodontic principles, further periodontal disease occurs. Pressures from various orthodontic appliances cause resorption of portions of the alveolus and the tooth root, and mobility of the affected teeth. Too rapid movement of the teeth, the moving of teeth into traumatic occlusion and the failure to allow a sufficient rest period between applications of orthodontic pressure—these are among the improper orthodontic procedures which may induce extensive alveolar resorption and eventual tooth loss.

The application of strong orthodontic forces produces a compression of the periodontal membrane, blockage of the capillary circulation and eventual tissue necrosis. In teeth which have been exposed to great stress, there have been observed pronglike cementum hyperplasias which provide firmer tooth anchorage. Bone and cementum resorption may be anticipated and do occur even though the resorption of cementum is not the rule. The rendering of orthodontic treatment has been held possible because of the greater resistance of the cementum to resorption. Oppenheim is not in agreement with this belief and as a matter of fact, states very emphatically that researchers are in error when they overemphasize the opinion that human cementum reacts less readily than bone to pressure by resorption.

Bone resorption occurs on the side of pressure and is apposed on the side of tension. The elimination of trauma through reduction of strong orthodontic forces allows for tissue changes incident to repair.

From his researches, Skillen indicates that a greater amount of rotation was obtained in five days with a lesser amount of force than was obtained in three days with a greater force. Further researches by Skillen showed the activity of bone resorption still present thirty days after removal of bands and ligatures when the latter appliances were causing gingival inflammation.

Breitner has investigated bone changes resulting from experimental orthodontic treatment, and has indicated that bone's biologic plasticity enables the orthodontist to move teeth without disrupting the latter's relations to the alveolar bone.

Oppenheim concludes that there should be applied only slight forces not in excess of that which the tissues can tolerate. He feels that orthodontic tooth movement depends upon bone resorption and formation which are in turn stimulated by properly regulated pressures and tensions.

It is not always simple to determine just how the tissues will react to orthodontic pressures because very often the responses of the periodontium are unexpected and unpredictable. Pressures which elicit optimum tissue responses in some cases will produce pernicious responses in others. The reaction to the same amount of force varies with each person depending on some individual and other unknown factors. Becks has indicated that excessive cementum resorption of the tooth root in careful orthodontic treatment may indicate the presence of a systemic disorder, possibly of the endocrine system.

2. ORTHODONTIC AND PERIODONTAL DISEASE IN CHILDHOOD

In orthodontics, growth and development are of primary interest to the operator since children throughout the entire period of growth are being considered. Because periodontal disease has been considered more or less a disease of adulthood and late adolescence, it has been felt that by and large many of the problems of growth and development are rarely causes for too much concern on the part of the periodontist. Until recently, therefore, the

study of periodontal disease in children has received relatively little emphasis. The efforts of McCall have been responsible for renewed interest in this most important phase of periodontal disease. Zappler is of the opinion that early detection, prevention and treatment of periodontal disease in children probably could lead to the near eradication of the disease in later life, and the production of a generation of adults comparatively free from the accumulated defects of early periodontal neglect. The fact does remain that acute periodontal disturbances in childhood are rather common. Chronic disturbances are less frequently seen, while degenerative changes are rare.

Among the factors which contribute to localized periodontitis is the malalignment of erupting teeth. It is here that orthodontic services are needed, and it is in such cases that there are produced areas of local gingivitis from improperly fitted orthodontic bands and springs or/and orthodontic appliances in unhygienic mouths. Other periodontal involvements of childhood are seen in those cases in which, during improper orthodontic treatment, movement of the teeth is not carefully supervised and a cusp to cusp tooth relationship occurs. This produces a *"kinetic traumatism"* which in turn is manifested clinically by tenderness, increased mobility, excessive tipping and ultimate loss of teeth.

The careless adjustments of bands and ligatures during orthodontic treatment may injure the periodontal attachment, just as these appliances may accidentally cut the soft tissues during placement, adjustment or removal. Loosened bands have been known to injure the soft tissues, and manipulation of the appliance by patients may result in the application of excessive forces which would seriously injure the periodontium.

3. PERIODONTAL DISEASE RESULTING FROM ORTHODONTIC CORRECTION OF NON-OCCLUSION

Non-occlusion is a condition in which one or more teeth cannot be brought into contact with their opponents in any position which the mandible may assume. Possibly the result of congenital disorders, disease, accidents, abnormal habits, faulty dental work or derangement of the growth-influencing factors during periods of tooth eruption, non-occlusion is recognised as one of the most influential of the periodontopathogenic factors. Inflammation of the gingivae and atrophic bone changes are most likely to follow the lack of functioning in these tissues unless artificial stimulation is employed.

The bringing of the teeth into occlusion may be accomplished by occlusal surface grinding, restorative procedures or by orthodontic correction. One or all of these methods are usually necessary. The choice of technics depends on the particular case and therefore requires great judgment and discretion on the part of the operator. Orthodontics is indicated if and where the conditions permit. All cases of nonocclusion are not amenable to orthodontic treatment, and further deterioration of the periodontium will certainly occur if such attempts are made to establish occlusion in certain cases.

C. ORTHODONTICS AND PERIODONTAL DISEASE THERAPY

1. REMOVING RESPONSIBLE FACTORS IN PERIODONTAL DISEASE BY ORTHODONTIC MEANS

The treatment of diseases of the periodontal tissues consists of three phases: These are (1) the removal of the responsible factors, (2) the elimination of symptoms and (3) the institution of measures for the preservation of oral health.

It is in the first and second of these phases that the orthodontist can be of especially great assistance to the periodontist. The correction of various forms of malocclusion contributary to periodontal disease, and the elimination of such symptoms as migration of teeth are the most outstanding areas in which orthodontic treatment is essential. Although such treatment can and does involve the move-

ment of several teeth, it is usually good periodontal practice to limit orthodontic intervention to simple procedures entailing the movement of single teeth into line to prevent occlusal trauma, and the closing of minute spaces to preclude vertical food impaction. The grassline ligature is often used to realign a drifting lateral incisor. This is a very effective method of utilizing a twisted silk cord as a temporary splint for ligation of movable teeth to firmly anchored teeth. The Hawley appliance with a spring arm, and the band techniques will also accomplish movement of single teeth. The tendency of these teeth to move back to their original places makes it necessary to secure retention with fixation by some form of splinting.

The treatment of crowded anterior teeth producing periodontal disease when the former occur in young individuals is usually an orthodontic problem, and the duty of the periodontist is mainly to aid the orthodontist by caring for the gingival tissues during treatment. When this crowding occurs in older individuals, the tooth most affected by periodontal disease is removed, a procedure which is usually sufficient to cause the other teeth to realign themselves. In those cases in which the latter does not occur, the grassline ligature is recommended to move the teeth into the required positions. Orthodontic intervention is necessary when there is a buckling of the teeth in both arches. This is also the case when there is the problem of a deep overbite. Much has been written about the bite plane to control these conditions. Hemley, Margolis, and Strang have done a great deal of work with this appliance and have reported favorably of its use. Hemley stated that after eleven weeks of use, mandibular incisors did not show evidence of either bone or cementum disfunction. The appliance consists of a full palatal plate which has a bite wall from cuspid to cuspid. The bite wall is articulated with the lower six anterior teeth so that these teeth strike the plane perpendicularly. The remainder of the lower teeth are without opposing occlusion when the mandible moves to occlude. These teeth elevate themselves and thus the bite is raised. The use of the bite plane is not contraindicated by periodontal afflictions of the lower incisor or cuspid regions as long as these teeth remain functional. It stimulates function for these teeth.

2. RESTORATION OF FUNCTION

The use of splints and stabilizers is a most necessary step in periodontal therapy. Teeth which have become loose through loss of support because of alveolar bone resorption, can be splinted together successfully. Wandering or drift of the teeth,—a common symptom of periodontosis—can be prevented if teeth are splinted together.

The fabrication of splints is a procedure which may be best accomplished by the crown and bridge prosthodontist. Certainly the variety and technique of mechanical construction which are demanded in certain cases, would require the expert assistance and guidance of an experienced prosthodontist.

Appliances composed of twenty-eight gauge steel ligature wire are among the more common temporary splints. Removable acrylic labio-lingual appliances, permanent removable and fixed splints, cast splints, soldered crown splints and pinlay bar splints are a few of the many ingenious devices accessible to the periodontist, and which—with the consultation and cooperation of both orthodontist and prosthodontist—can very often be utilized to aid in the successful treatment of particular periodontal cases.

SUMMARY

A great deal of work has been done on the effects produced in the normal periodontal tissues as a result of various orthodontic, traumatic, and other influences.

An attempt has been made to indicate those areas in which there are relationships between orthodontics and periodontology according to the following outline:

A. Orthodontics and Experimental Pathology in Periodontal Research
   1. Orthodontic Research
   2. Periodontology Research

B. Orthodontics and Periodontal Disease Etiology
   1. Incorrect Orthodontic Procedure
   2. Orthodontics and Periodontal Disease in Childhood
   3. Periodontal Disease resulting from Orthodontic correction of Non-occlusion

C. Orthodontics and Periodontal Disease Therapy
   1. Removing responsible factors in periodontal disease by orthodontic means
   2. Restoration of function

CONCLUSIONS

Orthodontics has been associated with the etiologic and therapeutic phases of periodontal disease. It has been listed as causing periodontal disease if improper orthodontic procedures are used, and it has also been listed as one of the most important of the treatment procedures for the maintenance of high oral hygiene.

It would appear that the incidence of periodontal disease resulting from incorrect orthodontic procedure can be drastically reduced and the number of cases of periodontal disease recognized as being amenable to orthodontic treatment materially increased if there would exist closer clinical, research, and professionally cooperative relationships between the orthodontist and periodontist. Recognition of the relationships which do and should exist is not enough. These must be implemented, so that true overall dental benefits to patients may be more nearly assured.

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Tissue Metabolism and Periodontal Disease*

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CONSIDERABLE progress has been made since the beginning of the century in the exploration of the nature of periodontal disease. Histopathological studies have been the major source of fundamental information. Valuable though the findings in histopathological research have been, its limitations are becoming increasingly apparent. If the interplay of local and constitutional factors in the etiology and progress of periodontal disease is to be thoroughly understood, it is necessary to determine the significance of the morphological changes it entails on a basic physico-chemical level. Recognition of the inadequacy of knowing only what tissues look like microscopically in periodontal disease and appreciating the importance of clarifying what the tissues are doing, pointed to the study of tissue metabolism in the periodontal region under physiological conditions as an initial step in the required direction.

Metabolism[1] may be defined as "the sum total of tissue activity as considered in terms of physico-chemical changes associated with and regulated by the availability, utilization and disposal of protein, fat, carbohydrate, minerals, water and the influences which the endocrines exert on these processes." Metabolism is concerned with the production of energy required for the maintenance of vital functions of individual cells and tissues. The following laboratory methods may be used in the study of tissue metabolism: chemical studies of tissue, histochemical study, the study of enzyme extracts, radioactive isotopes and the determination of oxygen consumption. Information obtained regarding the periodontal tissues in health and disease using the above methods has appeared in several reports.

Using chemical methods[2] the cholesterol, phospholipid, total lipid and cholesterol after saponification were determined in infant and adult gingival tissue. The phospholipid content of the gingiva lies between that of skin and intestinal mucosa—more nearly allied to mucosa. The total lipid value lies between skin and mucosa, resembling skin. The cholesterol content of gingiva is comparable to skin and slightly larger than mucosal values.

In normal and inflamed human gingival tissues phosphatase has been noted in the endothelial cells, in the capillary walls and possibly in the connective tissue fibers. No phosphatase was reported in the epithelium or in desquamated epithelial cells.[3] The amount of phosphatase in blood from the gingiva in cases of periodontal bone loss was reported as greater than the phosphatase content of the blood plasma. Cases of so-

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called rapid alveolar crest destruction showed a greater increase in the phosphatase content of the blood in the gingiva.[4] In fresh cadavers with evidence of bone destruction the phosphatase concentration in the areas of alveolar bone destruction was five times that in the tibia.[5]

The presence of glycogen was reported in normal human gingiva whereas pathological gingival tissue was described as markedly deficient in glycogen.[6] The glycogen content of surgically traumatized gingival tissue is reduced about one-half as much as in chronic gingival inflammation. Depletion of gingival glycogen was noted in experimental animals in alloxan diabetes whereas subjecting dogs to experimental trauma produced microscopic evidence of pressure changes in the periodontal tissues, but no alteration in glycogen.[8] In the saliva of individuals with periodontal disease the rate of protein putrefaction has been reported as more rapid than that occurring in the saliva of individuals with normal gingiva.[9,10] In another study[11] no significant difference was found between the glucose content of the blood from the gingiva and finger in patients with periodontal disease.

It has been suggested that the gingival biopsy could be of value in the diagnosis of secondary amyloid disease on the basis of its presence in 78 per cent of the cases of amyloidosis.[12] The amyloid is deposited extracellularly adjacent to the capillaries in the papillary layer of the gingiva, immediately subjacent to the basal layer of the epithelium, and appears as islands of amorphous hyalinized material which stains. metachromatically with crystal violet.[13]

Histochemical study[14] of gingival tissue using the Hotchkiss leucofuchsin periodic acid method[15] revealed the following aspects of normal gingiva obtained from patients of different ages: The boundary zone between the connective tissue and epithelium was formed by an optically homogenous "basement membrane" consisting of a ground substance of insoluble highly polymerized carbohydrate-protein complex. A ground substance consisting of a highly polymerized carbohydrate-protein complex and small amounts of glycoprotein residues and uronic acid derivatives formed a significant component of the gingival connective tissue. The staining of the connective tissue fibers was attributed to an intrinsic glycoprotein structure or a covering of glycoprotein ground substance. The cytoplasm of the connective tissue cells only infrequently contained glycogen or glycoprotein granules. The epithelial cells, particularly those of the middle third of the epithelial layer contained a carbohydrate material in the cytoplasm presumed to be glycogen. The epithelial cells were separated by a glycoprotein network.

Radioactive isotope study revealed the radioactive phosphorus content of maxillary bone to be considerably higher than that of mandibular bone. In addition, a greater deposition of P 32 in the dentine of the root than in the dentine of the crown was attributed to the existence of a system of canals in the cementum which permit the passage of fluids from the periodontal membrane to the dentine.

The literature related to the problem of metabolism of the periodontal tissues appears rather sparse in comparison with the abundance of information regarding other tissues of the body. Aside from the contributions noted above, consideration of the metabolic aspects of the periodontal tissues has, of necessity, has been limited to conjecture predicated upon information derived from tissues other than those of the periodontal region. Stimulating though such speculation may be, it hardly suffices as a dependable substitute for information regarding the metabolism of the periodontal tissues which would evolve from investigation of metabolic processes within the periodontal area itself.

The demonstrated usefulness of the Warburg Manometric Technic for the study of oxygen consumption of numerous body tissues[17-21] and an earlier preliminary report[21a] dealing with oxygen consumption in gingival disease suggested the advisability

of further investigation with this technic in the periodontal field.

As noted above the determination of oxygen consumption is one of the methods of evaluating the metabolic activity of tissues. In general, oxidation can be accomplished by the addition of oxygen, the removal of hydrogen, or by the withdrawal of electrons. With most metabolites the primary oxidation is effected by the removal of two hydrogen atoms or 2 hydrogen ions and 2 electrons.[21b] The available oxygen eventually reacts with the hydrogen to form H_2O or H_2O_2. Free energy is liberated by the oxidation of the hydrogen.

The sequence of changes entailed in oxidation in tissues is carried out by the operation of a series of enzymes and catalysts[22] where action is so coordinated that the degradation of the molecules of the metabolites proceeds in a step-like fashion. Smooth delivery of energy in a manner most advantageous to the cells is accomplished by the liberation of a certain portion of the total metabolite in each step of the oxidation process. Under physiological conditions the gingival tissues require energy for the maintenance of cellular viability and for the cellular activity essential for the preservation of a multilayered protective covering.

Using the Warburg Manometric Technic[23, 24] a study was conducted to determine: a. the oxygen consumption of normal human gingiva, b. whether oxygen consumption was altered in cases of chronic gingival inflammation, and c. whether such alterations could be correlated with microscopic gingival changes.

Eighty-six specimens of gingival tissue from 46 patients were studied. The tissue was removed with a scalpel or a scissors and placed immediately in a vial containing modified Ringer's solution (MPBR), buffered with phosphate (pH 7.5). A portion from each specimen was set aside for microscopic study. The specimens consisted of marginal gingiva (with the exception of 5 specimens of oral mucosa over edentulous alveolar ridges) which ranged from 5 to 50 mm. in length, 3 to 8 mm. in width, and 3 to 5 mm. in thickness. Represented in the specimens were normal gingivae and gingivae with varying degrees of chronic inflammation and enlargement taken from patients 13 to 60 years of age.

The gingival specimens were divided on a histopathological basis into 4 groups. 1. Normal—specimens which presented a minimal inflammatory proliferative or degenerative change. 2. Slight inflammation and proliferation—specimens which presented slight chronic inflammatory infiltration and/or proliferation of epithelium and connective tissue and new blood vessels. 3. Marked inflammation and proliferation—specimens which presented marked chronic inflammatory infiltration, and/or proliferation of epithelium and connective tissue and new blood vessels. 4. Marked degeneration—specimens in which degeneration and necrosis were the only or predominant findings.

Correlation of the QO_2 values with the underlying microscopic gingival changes revealed the following: The oxygen consumption of human gingiva is measurable by the Warburg manometric technic. The average oxygen consumption for normal gingivae was $1.6 \pm .37$. Oxygen consumption was essentially unaltered in gingivae with slight inflammatory and/or proliferative changes. There was a tendency toward increased oxygen consumption in gingivae with marked chronic inflammation and proliferation of connective tissue and epithelium. There was a tendency toward decreased oxygen consumption in gingivae which were essentially degenerated and necrotic and contained few, if any, viable tissue elements.

The normal QO_2 of $1.6 \pm .37$ obtained in this study for normal gingiva is comparable to that reported for human skin[17, 25] ($1.48 \pm .48$; 2.1 with variations between 1.0 and 3.6) and lower than that of liver[18] (guinea pig, 4.04) and kidney[18] (guinea pig, 13.0). The similarity in the oxygen consumption of human gingiva and skin

and the differences in oxygen consumption between gingiva and liver and kidney might be anticipated by a comparison of the functional activities of the respective tissues. The tendency toward increased oxygen consumption in marked chronic marginal gingivitis is apparently associated with an infiltration of inflammatory cells, the formation of new blood vessels and connective tissues fibers, and the proliferation of connective tissues and epithelium. It is of interest to note that the increased oxygen consumption associated with cellular activity in chronic inflammation in human gingiva is comparable to the QO_2 values reported with similar microscopic changes in other tissues (kidney,[19] tendon,[20] and skin[21]).

Demonstration of the applicability of the Warburg Technic for the determination of gingival oxygen consumption suggested that it might be used to augment existing information regarding gingival repair which had been derived solely from clinical and histological observations.[26, 27, 28] A study[29] was therefore conducted correlating oxygen consumption and histological changes in the various stages of healing following gingivectomy in the dog.

FINDINGS

The findings indicated that the QO_2 of the normal gingiva of the dog ($1.3 \pm .07$) is comparable to that of humans ($1.6 \pm .37$). The oxygen consumption of the gingiva in the course of healing following gingivectomy varied according to a pattern related to the histological changes. At the fifth day of healing an elevation in QO_2 was associated with an influx of inflammatory cells, proliferation of epithelium and the formation of new capillaries. Edema and degeneration present at this stage tended to depress the overall oxygen consumption. (An initial fall in oxygen consumption immediately after operation described in the skin,[30] could not be detected in the gingivae because the specimens obtained earlier than the fifth day were unsuitable for satisfactory QO_2 determination.) A further rise in QO_2 from the fifth to fourteenth day resulted from continued proliferation of epithelium, new capillary formation, fibroblastic activity, and a reduction in the inflammatory and degenerative changes. This was followed by a reduction in QO_2 from the fourteenth to the twenty-first day, at which time the pre-operation level was reached. There was an associated reduction in vascularity and cellularity of the connective tissue, absence of fibroblastic activity, resolution of inflammation and maturation of the epithelium. The pre-operation QO_2 level attained at the 21st day was maintained with slight variation throughout the remainder of the sixty-four day experimental period, in the course of which the microscopic appearance of the gingiva was essentially that observed before operation.

This study suggests that it is the nature of the tissue changes in the reparative process that determines the oxygen consumption and that it is not the oxygen supply which governs the sequence of cellular changes. In the Warburg Manometric Technic, the tissue is bathed in a medium saturated with oxygen so that the pressure gradient of oxygen between the medium and the tissue is at a maximum during the period when the tissue is metabolizing. The gingival specimens obtained at different stages of repair use different amounts of oxygen according to the metabolic requirements of the cellular components. This relationship of oxygen consumption to cellular activity exhibited by healing gingiva is in accord with the concept of Best and Taylor,[31] who note that, whereas increased activity of a tissue entails an increase in oxygen consumption, increase in the oxygen supply above the tissue requirements does not increase oxygen usage. The present findings, therefore, discourage the assumption that the artificial introduction of oxygen would hasten appreciably the sequence of cellular changes involved in gingival healing.

Normally a greater demand of tissues for oxygen is met by increasing the total blood flow through the tissues, thus main-

taining a high intracapillary oxygen pressure, and by raising the coefficient of oxygen utilization; that is increasing the quantity of oxygen abstracted from a given volume of blood.[31] A steep oxygen pressure gradient between plasma within the capillaries and the tissue cells increases the coefficient of oxygen utilization. Nature increases the pressure gradient by increasing the metabolic end products such as carbonic and lactic acids, by lowering the oxygen tension within the tissue cells, by causing a rise in the temperature of the tissue, and by shortening the distance through which the oxygen must diffuse; that is, by opening up more capillaries and thus reducing the radius of tissue supplied by each capillary. Our observations of healing gingivae revealed no microsopic evidence of circulatory inadequacy which might be construed as impairing the healing processes by interfering with the oxygen requirements of the tissues.

In another approach to the subject of gingival metabolism a study was conducted in our laboratory using the surface smear technic with the Shorr stain on the gingiva of 215 female patients. The purposes of this study were to determine whether there were any variations in the cellular activity of the stratified squamous epithelium of human gingivae which could be detected by this method and whether such variations were correlated with different phases of the menstrual cycle. The conclusions drawn from this study have been reported elsewhere in detail.[32] However, one aspect of the findings is pertinent to this presentation. It was observed that a notable trend toward diminished keratinization occurred in normal gingiva with increasing age. In addition, the diminished gingival keratinization which occurred in the menopausal and post-menopausal periods represented a late phase of the physiologic tendency toward diminished gingival keratinization which occurred with age.

It has been suggested[33] that under physiological conditions, epithelial cells tend to become cornified as the distance from the basal layer is increased. Diminished gingival keratinization with increasing age may therefore be considered as indicative of physiological atrophy of the gingival epithelium arising from reduced metabolic activity of the individual epithelial cells. Further exploration of the nature of this physiological process would be most informative and very likely constitute an effective background for the approach to many hitherto unexplored aspects of gingival disease.

In summary, the inadequacy of histopathological methods as the sole approach to the problem of periodontal disease has been noted. The need for information regarding the metabolism of the periodontal tissues in health and disease has been stressed. Findings in chemical, histochemical, radioactive isotope and oxygen consumption studies of the periodontal tissues have been reviewed. Attention is directed to the sparsity of available information regarding the details of metabolic changes which occur in the periodontal tissues in health and disease. The need for further metabolic studies of the periodontal structures, rather than speculation regarding metabolism in the periodontal region predicated upon findings in other tissues in the body has been stressed.

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Thanks are due to Dr. George Paffenbarger, Chairman of the Dental Study Section of the National Institutes of Health and the other members of his section, for releasing the preceding series of papers for publication in the Journal of Periodontology. The Section is to be commended for sponsoring this Conference, for it should prove to be a valuable stimulus to studies of periodontal diseases—and no one denies the need for a stimulus. It is hoped that similar conferences will be held periodically.

Announcements

Academy of Periodontology to Sponsor Essay Contest

Plans were approved at a recent meeting of the Council of the American Academy of Periodontology for two essay contests to be held for undergraduate dental students each year. Details of these contests have already been sent to the deans of the various dental schools in the United States and Canada. Briefly the rules are:

A. Local Contest. The best essay on some phase of Periodontia that is written each year by its undergraduate students is to be selected from each dental school in any manner that its faculty desires. The name of the winner is to be submitted to the Editor of the Journal of Periodontology before May 1 of each year, and the winner will receive a year's subscription to the JOURNAL OF PERIODONTOLOGY.

B. National Contest. The winner of each local contest is entitled to enter a national contest if he so desires. Papers are to be submitted to the Editor of the JOURNAL OF PERIODONTOLOGY before May 1 of each year. The Editor will then submit the papers to the Undergraduate and Postgraduate Committee of the Academy for judging. Annual prizes will be $50, $25, and $10.

It is hoped that this project will promote interest in periodontia in the dental schools. A few local contests were held last year even though details of the contest were not approved until late. Winners are as follows:

Baltimore College of Dental Surgery—
"Comparison of the Efficacy of Two Different Solutions in the Treatment of Acute Necrotizing Gingivitis"—Lloyd S. Noel and Henry W. Teyker.

Indiana University School of Dentistry—
"Hyperplastic Gingivitis"—Kenneth L. Smith.

Northwestern University Dental School—
"Dental Foci of Infection"—Hamilton Strawbridge Cloud.

The Second Annual Berkshire Conference in Periodontology and Oral Surgery

Tufts College Dental School will conduct the Second Annual Berkshire Conference in Periodontology and Oral Pathology at Eastover, a resort hotel in Lenox, Massachusetts, June 12 to 16, 1951. The conference will consist of four days of lectures, seminars, and panel discussions regarding clinical problems by outstanding teachers and clinicians in dentistry, medicine, and their related fields.

The curriculum is arranged so that the mornings and evenings are devoted to professional activities, with afternoons open for recreation. A special activities program is scheduled for the ladies. For further information regarding this conference write:

The Berkshire Conference
Tufts College Dental School
136 Harrison Avenue
Boston 11, Massachusetts

Editorials

Supply of Natural Bristle Toothbrushes Threatened

Before World War II it was generally conceded that bristles from the hides of animals living in cold climates were the best for toothbrushes. Most of the bristles came from the wild boar of North China. When the war interfered with this source of supply a nylon filament was developed and generally used. The first nylon bristles were very stiff, and inasmuch as they did not soften when moist as would a natural bristle, they were found to be too harsh by many patients. As a result many dentists are prejudiced against them, and recommend only natural bristles, believing that natural bristles are more resilient and more easily tolerated by gingival tissue.

On December 21, 1950, the National Production Authority issued an order (M-18) restricting the use of pig's and hog's bristles which will have a marked effect on the manufacture of toothbrushes. This order, which was modified on January 12, 1951, provides for the manufacture of only the following types of toothbrushes:[1]

1. Nylon, all textures.
2. A brush made with 70% natural bristle and 30% nylon.
3. Natural bristle, 100% taken from bristle not longer than 2⅝". This bristle length will make only a fair "Medium" or a Soft to Medium texture brush.
4. A toothbrush made with other substitute materials such as fibre, etc.

Of course the object of the NPA Order M-18 is to conserve bristles, and the dental profession will want to cooperate with all plans associated with defense. The fact that the eighteenth restriction order issued by the NPA deals with bristles emphasizes that a shortage will exist, particularly in the better imported bristles from China. However, it should be noted that of the 4,479,131 pounds of Chinese bristles which were imported in 1949, only a little over 1% (60,000) pounds were used in toothbrushes.[2] If the NPA order could be changed or interpreted to allow the manufacture of 70% of the quantity of last year's toothbrushes, the same saving of bristles will result and still make it possible for most patients who desire natural bristle brushes to purchase them. Dentists who prefer to have their patients use natural bristle brushes should

1. Millard, Glen (Lactona, Inc.)—Personal Communication.
2. Patterson, R. P. (Pycope', Inc.)—Personal Communication.

express their opinions to Mr. Ed. R. Killam, Industrial Operations Bureau, National Production Authority, Department of Commerce, Washington 25, D. C.

Unfortunately the combination of 30% nylon and 70% natural bristle is not feasible in toothbrushes; preliminary tests show that in such combinations the nylon bristles wear away more slowly and are soon left sticking up above the natural bristles. However, the manufacturing of 100% nylon bristle brushes has been so well perfected in recent years that restrictions of natural bristles will not prove to be nearly as crippling as it would have ten years ago. More and more dentists, including the writer, have found that the finer nylon bristles with a diameter of .010-.012 inch are stiff enough to be satisfactory and still not be irritating. Such brushes last longer, are easier kept clean, and are much more easily standardized in their stiffness. Since nylon bristles do not soften in water, however, patients should be instructed to use a brush with less stiff bristles than if they used a brush made of natural bristles.

Food for Thought

"Never have the achievements and prestige of the science of diet and nutrition been greater than now. With such success must go an equal measure of responsibility for continued progress."[1] To this we wish to add the thought that with the advancement of the science of nutrition goes the responsibility of *utilizing* the increased knowledge. Quoting further from a recent article by Dr. Keys,

"A . . . series of suggestions (regarding the influence of diet on degenerative diseases) comes from life insurance experience. An outstanding finding of the analysis of all such records is a direct relation between overweight and general mortality. Closer analysis indicates that the high mortality among relatively obese persons is primarily caused by high rates of hypertension, cancer, and the other degenerative diseases.

Further suggestions are provided by data from periods of undernutrition, particularly in the two world wars. The vital statistics are not good, and the complications are numerous, but such as it is, the evidence from many parts of the world tends in the same direction: famine and prolonged food shortages are associated with a relative decline in mortality from some degenerative diseases. Cowdry's suggestion, recently given out to the daily press, that starving Europe may exhibit less cancer in the future is at least conceivable.

Where is all of this leading us? In the case of at least some of the degenerative diseases, the inference is plainly that nutritional advantage may be in the direction of *less* rather than more. After all, it is reasonable to expect that the optimal diet for growth may be different from that for maintenance; it may even be that the very regimen which best promotes growth is the one which also promotes uncontrolled resumption of growth in adult tissues (cancer), or speeds up the whole course of later ontogeny (arteriosclerosis)."

This point of view gives one much food for thought!

1. Keys, Ancel. Influence of Diet ond Degenerative Diseases. Journal of the American Dietetic Association, 24:283, (April) 1948.

President's Address

By DICKSON G. BELL, *San Francisco, Calif. President 1949-50*

TODAY marks another milestone in the history of the American Academy of Periodontology.

It is my honor and pleasure, in behalf of the Officers and Members of the Council, to extend their greetings, and to welcome each of you who have assembled here in Atlantic City for this, the 34th Annual Meeting. To those who are unable to be here, we send our best wishes, and regret that they could not be with us. It is most gratifying to see so many present this morning, who have such a desire to extend their knowledge, that they are willing to travel long distances to participate in this three day discussion.

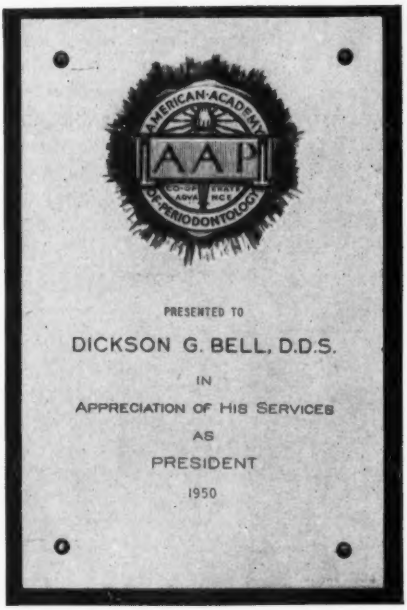
This tends to direct our thoughts to the first Academy Meeting and the conditions which existed when the five charter members met in Cleveland in 1914 to organize this Academy. They had vision, ideals, unity of thought and seriousness of purpose, as admirably expressed in the preamble of the Constitution which they adopted. Their objective was to advance dental science, especially the prevention and treatment of periodontal disease; to establish periodontia as a specialty of dentistry; to encourage investigations in the relationship of periodontia to general medicine; and to provide for the dissemination of this knowledge to the dental profession and the public.

It is interesting to review briefly the status of periodontia at that time. With the exception of a small group of men and women pioneers in this field, the dental profession paid little or no attention to the treatment of periodontal disease. Extraction of all such involved teeth was the general procedure, and the layman accepted the loss of his teeth as a natural consequence of middle age. There was no organized instruction in the dental schools—the teaching generally consisted of a few lectures and clinical demonstrations. The profession was somewhat skeptical of the clinicians who were attempting to approach the problem of prevention and therapy from a rational point of view. Some of the procedures would not be acceptable in the light of our present knowledge, while others, which are founded on a meager concept of histo-pathology and probably mostly empiricism, are still fundamental to the present day practice of periodontia.

Clinical periodontal practice in 1914 discloses some interesting facts and of course many differences of opinion. The preparation of the tissues before treatment received some attention at that time. Rinsing of the mouth with hot solutions and irrigation of the pockets were done to remove the debris and suppuration. Talbot's solution (iodoglycerol) was recommended to be applied to the teeth and gingival tissues to "destroy the bacteria." This was about the time when the use of vaccines was introduced. Injections of autogenous or stock vaccines in combination with local treatment of the lesion was believed by many to be the answer to their problem. Removal of vital and normal pulps was advocated on the theory that it had a therapeutic benefit by increasing the blood supply to the periodontal tissues.

It was the practice of some clinicians to introduce acids into the pockets to soften or loosen the hard deposits and thus facilitate the removal of the calculus from the root surfaces. There was a difference of opinion regarding treatment of the alveolar bone—some advised light curettement, others believed this was not necessary, while another idea was the removal of what was called "diseased bone" by the use of special bone-burs. Several different preparations were recommended immediately following curettage to contract the tissues. Ethyl borate gas (Dunlop oxygen therapy) was used by some to improve the oxygenation

Read at the thirty-fourth Annual Meeting of the American Academy of Periodontology, Oct. 26, 1950 in Atlantic City, N. J.



Plaque presented to President Bell at the Annual Banquet Oct. 27, 1950.

of the tissues. The protection of the blood clot was generally accepted and many had their own "sealing dressings" or used sandarac varnish for this purpose.

It is interesting to note that the correction of excessive stress was advocated, together with a warning that "reckless grinding of the cusps should be avoided." Also, that loose teeth should be supported either by temporary or permanent splints. In the field of prevention and mouth care, the names of D. D. Smith, Fones, Sarrazin and Adair were frequently mentioned as advocates of the soft toothbrush, whereas others recommended massaging the tissues with a hard toothbrush.

To most of you, the year 1914 means the beginning of World War I but it was also the era when a two room house on a half acre of ground could be rented for five dollars a month, when a French dinner could be had for seventy-five cents, and when a Ford Runabout could be purchased for five hundred and seventy-five dollars. Another historic event took place in Cleveland at the time the Academy was organized and one which has since cost motorists money, developed many bad tempers and caused high blood pressure. The first red and green traffic signals in the United States were installed there in 1914. It was this Academy which gave the first green light to Periodontology.

In these past thirty-six years, much advancement has been made and our concept of periodontal disease has been changed as a result of the research work of the many investigators. There is a better understanding of the histology of the periodontal tissues and histo-pathologic changes that occur in the development of the lesion. The importance of oral diagnosis has been recognized and, with it, a better knowledge of the influence of the systemic factors. The development of standardized roentgenographic technics has provided a valuable agent in the diagnosis, treatment planning, and prognosis of periodontal diesase. In clinical procedures the advancement has not been so rapid, but some progress has been made in working out the indications for treatment of the various types of lesions on a more rational basis.

During this period, the Academy has exerted its influence and its members have been a vital and potent factor in the advancement of periodontia to the point it now enjoys in the field of dental science. It has always had within its membership many of the outstanding clinical and research workers in periodontology. It initiated the organization of the American Board of Periodontology. The Academy was also instrumental in obtaining a separate section for periodontia at the meetings of the A.D.A., and its members have always taken an active part in its proceedings. The Academy has every reason to be proud that, in a large measure, the objectives of the organization have been maintained. This was inevitable, the calibre of membership being what it is, and is the expression of the role these members have played in the transition

of dentistry from a craft to a science of the healing arts. The scientific advancement of dentistry in the past half century has had a profound effect on dental education and the Academy contributed much to the progress that Periodontology has made during this period.

We look forward to the future with some uneasiness because of the present political trend of thought, nevertheless, with determination and courage and with the hope that right thinking will prevail and the autonomy of dentistry will be maintained. Continuous progress is necessary for the vitality of any science or group of scientists. The dental profession looks to the American Academy of Periodontology for that leadership in the field of Periodontology, which fact should provide the stimulus to still greater accomplishments. This is the challenge of the future.

All of us are aware of the fact that the JOURNAL OF PERIODONTOLOGY is recognized as one of the outstanding publications in the field of Dental Journalism. This is due largely to the zeal and untiring efforts of its Editor for twenty years—Dr. Grace Rogers Spalding. Last year, Dr. Maynard K. Hine took over this arduous task and has most capably continued the policy and high quality of this publication. The Academy is proud of its Journal and greatly appreciates the efforts of its new Editor. However, there is another way of expressing our sentiments and one which probably means much to an Editor—that is, your support of the Journal.

It was stated that one of the objectives of the Academy is to enhance the knowledge of the general practitioner in the prevention and treatment of periodontal disease. The Journal is one of the vehicles by which proper information in this special field is presented to the dental profession. Each of us has a responsibility in this regard and each should bring to the attention of the general dentist the practical benefits he will gain from reading such literature.

The Journal Subscriptions Committee has been most active during the past year in reminding the members of the Academy of this obligation. As a result of their efforts and what each of you has done individually, the subscriptions to the Journal have been greatly increased during this period. Dr. Romine and his committee have earned the commendation of every Academy member for their outstanding accomplishment. This was one of our objectives, and I personally appreciate what each of you has done.

The Academy has long realized the need for a classification of periodontal diseases that would meet with the general approval of all the members. A Committee was appointed two years ago and group discussions of this subject have been presented at the Round Table Luncheons. To evolve a plan of classification it is necessary to study the basic principles of etiology and pathology and make these applicable to the clinical picture. It is extremely difficult for the clinician to evaluate a classification based entirely on pathology and equally difficult for the pathologist to classify disease entities on the clinical findings alone. Therefore, if a classification of periodontal diseases is to be a useful tool to all concerned, it must be constructed from both points of view.

The Classification Committee has worked hard and diligently this year to present a classification and consequently has stimulated an increased interest. As a result, many different aspects and some differences of opinion have developed. The plan presented to the Council by the Nomenclature Committee should provide the solution to this problem, since it offers the opportunity and sufficient time for a thorough and frank discussion of all sides of the question. The efforts and splendid work of the Nomenclature and Classification Committees this year are greatly appreciated.

This Academy has now reached that stage of maturity when it should give serious thought to its plans for the future. Shall it continue as in the past years or has not the time arrived when it should care-

fully review the question of new members and their qualifications? The Committee on Constitution and By-Laws has made several recommendations but should we not "raise our sights" somewhat higher? The present membership is 331 and with 49 names to be voted on will make a total of 380 at the conclusion of this meeting. If the addition of at least 45 new members each year is continued, and there is every indication that it is likely to increase rather than decrease, the Academy membership in ten years would be over 800.

A continuation of the present plan could create a situation in the future where the Academy would be in competition with the A.D.A. Section on Periodontia. It would be just another Dental Society. A large membership frequently creates many problems and fosters groups or cliques, such as often exist in large organizations. I am certain none of you would like to see such a situation develop in this Academy. Our scientific meetings are now open to all non-members who are interested in this subject and its related phases and there is no reason for any change in this procedure. The opportunity is thereby provided for the general practitioner to be encouraged and educated in the prevention and treatment of periodontal disease. Thus our objective to serve the profession and public is maintained.

The name of this organization is the Academy of Periodontology. An Academy is an association or society of men eminent in literature, science and art—in this case, men and women whose purpose should be the advanced study of periodontology. In past years, the Academy has devoted much of its efforts, successfully let me add, to stimulating an interest in periodontia. The dental profession and the public are now periodontally minded. Also, it has done much toward educating many of its new members in the fundamentals of the subject. This basic teaching is now being given in the Dental Schools through undergraduate study of periodontology and many of them have established separate Divisions of Periodontia. University extension courses and post-graduate work in this field are also being offered to the profession. The successful future of the Academy lies not in the size of its membership but depends rather upon those new members who come into the Academy already trained in the essentials and who have extensive practical experience—not necessarily those who practice periodontia exclusively but who devote the major part of their time to it, research workers and teachers of periodontology.

It may be well to consider further increasing the qualifications for new active members—and should they not be diplomates of the American Board of Periodontology and should they not devote at least two-thirds of their practice to periodontia? Exceptions might be teachers and research workers in periodontology and related subjects. The category of new associate members could well be evaluated and studied.

This matter of rapidly increasing membership is not a serious problem at present so long as the objectives and ideals of the Academy are maintained. It is brought to your attention at this time for consideration of a situation which is certain to develop in the future because of the increasing interest in periodontia. Is it the purpose and desire of this Academy to have a much larger organization? This is a question which requires serious discussion and deliberation now rather than five years hence.

There is a group, the Past Presidents, to whom you have given the highest honor this Academy has to offer. Without any reflection on their ages, we may call them the "elder statesmen." They have an understanding and knowledge of the workings and objectives of the Academy probably more than any other member, except possibly the Secretary. A Past President has had at least six years of continuous experience as a member of the Council, then as President-Elect, as President, and finally as Chairman of the Council. It is recommended that the Past Presidents act as a Committee whose function will be to make

a thorough study of the membership situation and report their recommendations at our next annual meeting.

There are occasions during the year when some question arises which requires official action, and which unless a meeting of the Council can be called, must be settled by polling the members of the Council by letter or else deferred until the next semi-annual meeting. Therefore, in order to expedite the disposition of such matters, it is also recommended that an Ad-Interim Committee should be added to the list of Standing Committees. It is suggested that the Ad-Interim Committee consist of the Chairman, the Secretary, and any three members of the Council. When, in the opinion of the Chairman or Secretary, a meeting of the entire Council is not possible, it should be the duty of this Committee to govern and transact the business of the Academy during the intervals between its regular meetings. It should meet at the call of the Chairman or any three members of the Council.

In conclusion, I wish to thank you for having honored me with the Presidency of the American Academy of Periodontology. I have thoroughly enjoyed working with the various Committees, the Officers and members of the Council. The successful functioning of the Academy during this past year, and whatever accomplishments have been attained, are due entirely to their unselfish and untiring efforts. The reports which were presented this morning gave evidence of this fact. I wish to take this opportunity to express my appreciation to each one of them.

There is one member of the official family who has a great responsibility as well as an enormous amount of work. His duties have multiplied greatly since the membership has increased. The Academy is most fortunate in having such an able and efficient Secretary. My thanks to Dr. Clarke Chamberlain for his guidance and assistance during the past year.

The success of this annual meeting is the result of thought, careful planning, and hard work on the part of the different Chairmen and their Committee members. It is an excellent example of co-operation and harmonious teamwork. It would be difficult to single out any one Committee for special words of commendation; to each of them—the Committee on Arrangements, Program, Scientific Investigation, Clinics, Round Table Luncheon, Entertainment, and Publicity, my thanks for the successful accomplishment of their assigned duties.

In the year that is ahead of us, I am certain the Academy, under the able leadership of my successor, will widen the scope of its influence and re-dedicate itself to the purpose for which it was founded.

Periodontist Appointed Dean

Members of the American Academy of Periodontology will be interested to learn that Dr. Harry Lyons was appointed Dean of the School of Dentistry of the Medical College of Virginia on January 1, 1951. Congratulations and best wishes to Dr. Lyons!

Discussion of Dr. E. R. Granger's Article

"Bio-Mechanics of Periodontal Disease"

By BALINT ORBAN AND HARRY SICHER*

We have read the article "Bio-mechanics of Periodontal Disease" by E. R. Granger with a feeling of despair. After an introduction with which we can heartily agree, the disappointment is just so much stronger, because it is soon clear that the writer can, not only not contribute anything to the establishment of a biomechanical approach, but that he causes great damage to the prestige of clinical and scientific dentistry. How can anyone try to help the standing of dentists by stating: "While we know comparatively little about the mechanism of the endocrine glands, fortunately, as dentists, we do not need to know." The first part of this statement is obviously not true and does not the second part mean to foul one's own nest?

"Bacteria are not the cause of dental disease." Does such a monumental sentence have a place in a scientific (?) paper? And what about the almost funny reversal of all we know that "the loss of bone and the formation of the pocket were what brought about the local irritating factor"? Is it really necessary to ask the dentist whether he has "ever stopped to wonder just exactly why" it is true "that a very important determining factor is the amount of bone which is left around the tooth"?

When Granger writes about the autonomic nervous system, he betrays a total lack of knowledge and understanding. "The autonomic nervous system is in turn controlled by the glands of internal secretion. Witness, for example, the excess growth of tissue brought about by overstimulation of the parasympathetic nervous system by excessive thyroid activity." It is exactly the *sympathetic* component of the visceral nerves that causes in [illegible] goiter the wide lid fissure, the wid[illegible] pupils, the accelerated pulse rate, the pro[illegible]eating and other symptoms. Which org[illegible]to overgrow in this disease? Who showed that "it has for example, been possible experimentally to halt the progress of early periodontal disease by stimulation of the parasympathetic nervous system" and how was this done? Who has shown that "it has been possible experimentally to cause local growth of tissue by continuous local stimulation of the parasympathetic nerve to that part." Also the statement that "the sympathetic nervous system controls the destructive processes involved in utilization of tissues; the parasympathetic nervous system controls the growth and repair of these same tissues," is by no means solidly founded. Best and Taylor* write about the general effects of the autonomic system as follows:

"Taken as a whole, the actions of the sympathetic division and its ally, the adrenal gland (sympathoadrenal system) are directed toward strengthening an animal's defenses against the various dangers which beset it, e.g., extremes of temperatures, deprivation of water or the attacks of its enemies."

"It is evident that the sympathectomized animal could not fend for itself, and, in the struggle for existence, would soon succumb to the hazards of the environment."

"Parasympathetic effects, rather than being characterized by a diffuse outburst of activity, as may result from sympathetic stimulation, are more localized in character. It has also been suggested that they are concerned with conservative and restorative processes, and the sympathetic with processes involving the expenditure of energy. Inhibition of the heart, contraction of the pupil for the protection of the eye from intense light and the activities of the digestive tract, through which the energy stores of the body are replenished, are frequently given as examples of acts of con-

1. J. of Periodon. 21:98, 1950.

*Physiological Basis of Medical Practice. 4th Edition, Williams & Wilkins, 1945.

servation presided over by the parasympathetic. These apparent differences in the activities of the two divisions have led some to speak of the functions of the sympathetic and parasympathetic divisions as *catabolic* and *anabolic*, respectively. It is an interesting but perhaps a too speculative generalization."

Did the author not feel that his statement: "then surely we must see that our real dental problem does not lie in the field of medicine but rather in the mechanics of function" contradicts his own plan to find a new "philosophy" for dentistry, based on a biological approach?

He writes: "In all truth, dentistry, to be completely successful, requires a philosophy *more* than a technique." We would suggest that dentistry, most of all, needs better-educated dentists.

DISCUSSION OF DOCTORS ORBAN AND SICHER'S DISCUSSION

By Ernest R. Granger

The reaction of Drs. Orban and Sicher is neither surprising nor unexpected. Thank you so much for giving me the opportunity of answering it. It appears that their letter was written more in rancor than cold analysis. They either do not, or do not want to, understand the paper. If they did, then surely they would see that it does not reverse, but on the contrary complements and explains what they refer to as "all we know."

In attempting to answer them, I face the same dilemma that repeatedly arises in similar discussions. Since they have both made it abundantly clear in their own writings that they have no real understanding of oral physiology, they obviously would not be in a position to appreciate its significance, or the part it plays in periodontal disease. And by that same token it becomes difficult to carry on an intelligent discussion without such a common basis of understanding.

In that respect it reminds me of the situation with Dr. Orban's former preceptor, the late Dr. Gottleib, shortly before his death. After a full day spent in examination and discussion of the material upon which the paper is based, his own words were "To think that I could have spent all these years condemning the role of function in periodontia, without ever really understanding function and cusps. How is it possible this could have escaped me, and is not more widely understood?" His parting words, in the presence of one of Dr. Orban's good friends, were these, "You can tell anyone who asks you, I still believe that, as widely practiced, functional treatment does not aid periodontal treatment, but I now realize that is because they are not treating function at all, they are only maltreating it. I now realize that it is basically a functional problem, and can see the explanations for many of the puzzling things that we have all seen." This is the first time I have had occasion to quote him, but it is well documented by one of Dr. Orban's associates. At that time he expressed his intention to make extensive changes in his book, but unfortunately the amount he was able to write, which he sent to me for examination, was woefully inadequate. Just about enough to show that he did comprehend the problem.

In quoting passages from the paper, I am afraid that Drs. Orban and Sicher have fallen victims to the very thing I had in mind at the close of the paper when I asked that they look at the woods, not the trees. They have failed to grasp the whole picture. This does not contradict their local treatment, it merely complements it. For example, they have quoted a part of the sentence "Bacteria are not the cause of periodontal disease," without the rest of it, "In the mouth bacteria produce the symptoms which we call periodontal disease." i.e., the inflammatory process. The bacteria associated with periodontal disease are found in healthy mouths of all ages. If the bacteria per se caused the periodontal disease, then we would all have it. Something else must happen first to permit the bacteria to wreak their havoc. That does not mean that we should not treat the in-

fections when present, but it does mean that we should recognize the fact that it is only symptomatic treatment and go further.

They take particular exception to my statement that "While we know comparatively little about the endocrine glands, fortunately *as dentists* we do not need to know." I wish they had continued, "That is not *directly* our problem, we could not by that means *alone* produce a healthy mouth." That of course is the theme of the whole paper and an adequate answer would take more than these few words. In fact, it points up the problem of any paper dealing with as large a subject as this, the space limitations of how much can be said and the need of reducing it to an absolute minimum in the hope that the understanding of the reader will fill the gaps.

I am neither an endocrinologist nor a neurologist, nor have I ever laid any claim to being a scientist. I am a practicing dentist whose sole interest lies in treating as best I can patients with practical problems. My sole reason for giving any paper is that I have always been glad when requested to pass on any information I possess which may be of any interest or value to others in a similar endeavor, since that is the way I have acquired most of my own knowledge. Since I know that I cannot become an expert in endocrinology, I accept the work of medical men who have spent their lives in this field and are associated with research in leading recognized medical schools. My reason for saying "While we know comparatively little about the endocrine glands, fortunately *as dentists* we do not need to know," was based upon such a contact, and for very good reasons. During the course of developing this material, I had occasion to discuss it with a medical man engaged in this research at a leading medical school. His reply was, "We realize today that in the field of endocrinology, we have only scratched the surface. The one thing we do know, is how little we know, and as a dentist if you attempted to treat the endocrine problem you would be playing with fire." Under such circumstances I do not believe endocrinology lies in my field, since I cannot do anything in a practical way about it, and am not going to waste my time over it when there are so many more important practical measures that need more study. As I stated it does interest me only insofar as it helps me to understand my *dental* problem, and what I must do in a practical way to treat it within the confines of my own field and knowledge. As I said before, that of course is the theme of the whole paper.

I am afraid that it is very necessary to ask the dentist if he has "ever stopped to wonder just why it is true that a very important determining factor is the amount of bone left around the tooth," for the reasons stated in the paper. It is the proof that this is a functional problem, and endocrinology is the explanation of how. I am glad that Drs. Orban and Sicher find humor in the statement that the loss of bone precedes and causes the formation of the pocket. I know quite a few members of your own academy who have come to just such a conclusion from practical observations, in fact, the belief has in recent years become quite widespread.

My choice of words in discussing the relation of the autonomic nervous system seems to be quite a source of irritation. Since this is based upon personal conversations with men in this field (there is a great dearth of written material on it), it could well be that there is some mixup in terminology. Again, as a dentist it is of academic interest to me, but I am not interested in whether the words used are correct, but in whether the mechanism of their interrelated action explains my dental problem and treatment. And the best answer to that lies in the fact that it works. This may not be kind, and I have never stated it publicly, but a good part of my practice is composed of patients (including other dentists) who have been treated by recognized periodontists and face the loss of their teeth. The fact that this has worked so successfully in so many cases which were

diagnosed as hopeless would indicate that its application would lead to greater success in all cases. I suppose that depends upon your definition of success. My own definition of it as far as periodontia is concerned is that I do not consider a case successfully treated until nature has brought about a spontaneous improvement in the tissues of the mouth, and maintains it thereafter by normal metabolic processes.

That does not imply that all cases treated by local means are a failure. It does mean that these same mouths could maintain themselves longer if the whole problem were treated. It also recognizes the fact that there are some cases in which the metabolism is so low that no means can completely overcome it, but at least the rate of destruction can be slowed down. That being the case, can any periodontal treatment be called complete which does not also include the functional problem? Whether the question of how metabolism and function are related is correctly stated or not, there is no doubt that they *are* related. What about occupational disease, and its dental parallel, which Drs. Orban and Sicher so carefully ignored?

I am afraid that they have completely missed the point of the whole paper when they see a contradiction between the mechanics of function, and a biologic approach. The relation between the mechanics of function and the health of the tissues makes it a biologic necessity to health when malfunction is too great for metabolism to cope with. Does the orthopedic surgeon when he fits children with special shoes to improve the function of the bones of the foot and leg, lack a biologic approach? Or the cardiologist when he uses a mechanical electrocardiograph lack a biologic approach?

Lastly, I heartily agree with the closing statement, what dentistry needs is better educated dentists, but I would add, better educated in dentistry. A man who does not understand oral physiology is hardly in a position to discuss function.

Announcements

Invitation to the XIIth Congress of the International Arpa Madrid, March 13th to 18th, 1951

The XIIth Congress of the International Arpa will take place from 13th to 18th March, 1951, in Madrid, i.e., the week preceding Palm-Sunday. Tuesday 13th, Wednesday 14th and Thursday 15th of March, will be devoted to scientific reports and films concerning Periodontal disease.

The committees of the Spanish Stomatological Society and the Spanish Arpa will organize on Friday 16th a visit to the Escurial and on Saturday 17th a trip to Toledo. One the evening of the 17th there will be the official dinner followed by national festivities.

The participants could therefore have the occasion of visiting Spain during the Easter week which offers a particularly interesting and picturesque opportunity.

The scientific reports shall be announced to Prof. A. J. Held, Institut de Medecine dentaire l'Universite de Geneve (Switzerland), rue Micheli du Crest 43.

Principal topics:

1) Internal Medecine and Periodontal disease.
2) Prophylaxis and functional treatment.
3) Films on Periodontology.
4) Discussion of the topics which shall be submitted by the section of Periodontal disease at the XIth Congress of the F.D.I., London 1952.

Notes

This issue begins the second volume under the present editor's direction, and the occasion calls for a few comments. First of all, it is more than ever apparent that the first editor, Dr. Grace Rogers Spalding deserves all the credit and honor that the Academy can give; only by working with the Journal can one realize how much time and energy she must have spent in organizing and then editing the first twenty volumes. We are asking the Council of the Academy to consider changing the wording of the acknowledgement to Dr. Gillette Hayden on the inside cover of the Journal to include "Dr. Grace's" name. This is little enough to do, and certainly would not detract any from the honor paid to Dr. Hayden.

We wish to thank the many readers who have expressed their opinions about the Journal in recent months. No editor likes to be ignored; both praise and adverse comments are welcome since they indicate the publication is being read. Even good editors need some guidance, and we beginners need much help. Incidentally, thanks to the many readers who sent Christmas cards. We finally chose the one of Dr. Edgar Coolidge (Chicago) as the most dignified, Dr. James Holler (San Antonio) most decorative, Dr. Donald Derrick (London) the most unusual, although the card of Capt. Alling (Stuttgart) was a close second. Dr. William Crawford (Minneapolis) made the best photographs of his children, and we declared a tie for original photographs between Dr. Howard Hartman (Cleveland) and Dr. Balint Orban (Colorado Springs).

Dr. Orban of Colorado Springs, Colorado, announces the following courses are scheduled as follows:

1. Histopathology of the Oral Cavity (Microscopic) March 19-24, 1951.
   (Dr. J. P. Weinman and Dr. Orban instructing) ..... Fee $250.00
2. Histopathology of the Supporting Tissues
   June 11-16, 1951 ..... Fee $400.00
3. Theory and Practice of Periodontia
   June 25-30 ..... Fee $200.00

These courses have been approved by the Veteran's Administration.

(Continued from page 8)

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To the Editor:

I write to express my appreciation of the editorial "Preventive Care vs. Heroic Therapy" in the October 1950 issue of The Journal of Periodontology.

More of the same could well be the habit in the Journal since so much material is given to treatment and therapy.

To me it seems a shame that the dentists, whose only excuse for being is to minister to the health of the public, should have so little interest in the service of oral prophylaxis that this service is so frequently delegated to a hygienist.

There is no other means by which a dentist can learn so much about the existing conditions of a patient's mouth as during the careful observation while performing prophylaxis, correlated of course with the use of all other known aids to diagnosis.

As in so many phases of life and education, it is a thankless job and an uphill struggle to inculcate basic principles, but unless some persons in position to be heard lead in the campaign, who will do it effectively?

Again thanking you, I am

Fraternally,
Frank L. Adams